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THE WAR COLLECTION*

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TEN years have passed since, in the preface to a modest book which I ventured to write concerning war injuries of a portion of the human body in which I have always entertained a special surgical interest, use was made of the following words: "The present state of international unrest and the anxieties engendered by menacing war clouds have suggested that a small work with illustrations depicting war injuries, etc., might be timely". The threatening clouds that menaced Europe in 1939 burst a week or two before the publication of that small manual and plunged the world in a blood-bath: I trust that history will not repeat itself and that no such catastrophic denouement will synchronize with the appearance of this lecture in its printed form; yet the crust of civilization wears thin to-day and underneath are audible the mutterings of primeval fires, and the words from Lucan's *Pharsalia* which I quoted a decade ago, alas! keep ringing in my ears,

. . . quid in arma furentem
Impulerit populum, quid pacem excusserit orbi.†

No longer, it is true, is there clamorously proclaimed that specious doctrine of racial purity, the exaltation of one breed as the favourite of the gods, of which much was heard before the outbreak of the last Armageddon; no longer is there the loud and raucous praise of war as a natural and wholesome concomitant of a nation's life; the hideous Nazi Moloch has been laid low.

Although the war spectre may have altered its precise geographical setting to-day, its menace is still declared in the so-called 'cold war' which emanates from behind the Iron Curtain; and everywhere Communism,

multas per gentes et multa per æquora vectus,

rears its bestial, threatening head.

Despite the pious hopes of war-weary humanity, despite the desires of politicians and the diplomacy of Cabinet Councils, there remains not a shadow of doubt that so long as the earth is inhabited by the human race disease and war shall not cease. Nothing that has happened in the world's history during the 2700 years since Micah's time affords any cause for confidence in such idyllic Utopian bliss as that foretold to Micah the Moraschite in the days of Jotham, Ahaz, and Hezekiah, Kings of Judah: "they shall beat their swords into ploughshares, and their spears into pruning hooks . . . and they shall sit everyman under his own vine and under his fig tree; and none shall make them afraid". These ideals are not for pursuit by the realist whose aim is properly the Tacitean *securi pro salute*—without any fear as to safety—and failure on the part of those who control the helm of State to recognize that the best way to secure peace is to be prepared for war deserves and demands their impeachment and not sympathetic condonation for their credulity and folly. International wisdom is best shown by those whose conduct of affairs subserves the motto of the Cliffords, *Semper Paratus*, or that which underlies the escutcheon of the Gordon clan—*Animo non astutia*—courage, not craft.

Perchance the present state of unrest may be no more than the deadly 'disordering' which Thucydides noted as a sequel to the most triumphant campaign, yet this present time seems not altogether inopportune to direct men's attention to the War Collection, the *spolia chirurgica* of the late conflict, housed within the walls of the College.

But another reason, apart from the international situation, has influenced my choice of a subject for this Harveian Lecture. Long overdue is some public acknowledgement to the Medical Research Council for all it has contributed towards organizing such a collection as we possess here to-day. Twice in a quarter of a century has the Medical Research Council been approached for aid in this direction, and twice has it nobly responded and shouldered the heavy expense entailed up to this present moment.

The first War Collection underwent complete annihilation when this College suffered so severely on May 10, 1941, and a fresh effort had to be made from the very beginning. There remain, of course, scattered in many museums of pathology specimens illustrating injuries incurred in warfare, some dating from the Napoleonic wars, especially the Peninsular campaign and Waterloo. Few of these specimens do much to illustrate pathology, and most of them are little more than curious examples of the vagaries of missiles; some are also interesting inasmuch as they recall the names of famous military surgeons.

* Being the Harveian Lecture delivered within the precincts of the Royal College of Surgeons of England, May 25, 1949.

† Why did a maddened people rush to arms
And rob the world of peace?
(Lucan's *Pharsalia*, Vol. I, Bk. 1, 68, 69. Sir Edward Ridley's translation.)

Specimens from the Crimean War and the Franco-German War are mainly macerated bones showing fractures produced by bullets: even from the South African campaign such soft specimens as are available were obtained by personal effort.

The present War Collection is the achievement of many workers, who have organized, collected, transmitted, arranged, or mounted according to their opportunity or zeal. But a teaching collection such as this purports to be must be comprehensive; not only must the shelves be stocked with specimens exhibiting the ghastly results of the missiles, mechanisms, and machinery of 'total war', but expert photographic representation of these injuries is also required, as well as of the diseases incidental to war; the realistic touches of the artist's pencil and brush have also their place in recording traumatic incidents first hand, and through the medium of the illustrator's art the specimens round yonder walls are able to be studied by those whom distance separates from Lincoln's Inn Fields. The presence of illustrations as part and parcel of the War Collection is in the best Hunterian tradition, for in the original Hunterian Collection there were a great many fine drawings and pictures of pathological and other conditions, and during the years of the existence of the Museum a very large number has been added. It may be safely affirmed that the illustrations, colour photographs, etc., relating to the injuries and diseases incidental to the recent conflict constitute by no means the least valuable feature of the collection.

Grey Turner, in his Hunterian oration, reminded us that to collect is to follow a natural instinct: nevertheless, the acquisitive or possessive spirit and the motive to collect must vary in degree and in direction in different individuals and corporations: thus Sir Walter Scott's zeal included in its embrace ballads, old books, pictures, armour, and "auld knicknackets"; Charles Saint's penchant is for clocks, while others, according to the dictates of fancy, crave pottery, pewter, or porcelain, stamps, silver, or snuff-boxes. The older surgeons even before Hunter's day prided themselves on their anatomical and pathological collections, which they employed to illustrate their lectures; and if the "magna imago" of John Hunter could have returned to earth in 1942 he would indeed have been puzzled and amazed at the paucity of pathological trophies representing at that period three years of war. Possibly the exuberant zeal so manifest in the surgeons of the First World War to collect and transmit to a pathological centre specimens exhibiting the results of enemy missiles or the triumphs of their own art may not have been so conspicuous during the long years of this more recent conflict, but in fairness it must be stressed that the long lines of communication and the mobility of the front, especially in the Desert, did not favour the easy transport of specimens, nor was it likely that the latter would necessarily arrive at the Base in a condition that warranted permanent preservation for exhibition purposes.

Despite memoranda distributed to various E.M.S. officials and to selected medical officers and hospitals of the Fighting Services, and in spite of appeals through the medical press and the valuable *Bulletin of War Medicine*, material response was at first meagre in the extreme. It is true that some specimens, ultimately to prove of little value for museum purposes, had been collected in the Middle East, but after nearly three years of war the aggregate of specimens in this land scarcely reached double figures.

The war taught many the importance of close personal contact if some objective were to be attained, and it gradually became apparent to the Sub-Committee on Pathological Specimens of the War Wounds Committee set up by the Medical Research Council that something further was required than merely to rely upon the goodwill of surgeons and pathologists in sending material to the collection centres which had been arranged. The need seemed to be established for an enthusiastic, energetic pathologist who would tour Britain region by region, gathering specimens and personally securing the co-operation of surgeons and pathologists, and stimulating them to collect specimens against the visitor's next call. An entrée to all medical units in Britain was readily obtained, including those of the Fighting Forces. The value of personal contact between those who were more interested in the amount of the harvest and those to whom the precious opportunity of gleaning was vouchsafed, was confirmed by the precise regional origin of the specimens obtained as the result of enemy activity over this land or incidental to the activities of troops in training. The truth emerged that few specimens seemed to emanate from any area in Britain that had not been visited by Dr. Joan Ross or myself. Some of the best specimens in the Collection were, in fact, obtained in Great Britain, where a regular mechanism for the rapid transport of specimens from the periphery to the Central Pathological Office became well organized. Those surgeons whose sphere of activity was determined in the South-East corner of England were, I believe, perhaps the most consistently 'specimen-conscious' of all.

Collectors are of various kinds, and their welcome is wont to depend on the direction taken by the objects which it is their function to convey or transport: thus the dustmen and sanitary collectors of boroughs and rural districts have a popularity that may be the envy of tax-collectors or ticket-collectors in the corridors of trains, although in the case of each the traffic is 'outward-bound'. The popularity or unpopularity of the 'body-snatcher' of a former generation also varied according to the viewpoint of the surgeon-anatomist and the relatives of the deceased. Yet the tasks of a collector may not be without their humorous side: on one occasion during the war years the writer travelled by train from an intermediate wayside station near a Service Hospital, carrying with him one-third of a man with a right-sided diaphragmatic hernia and another package containing an amputated lower limb. While the luckless Surgeon Consultant had to pass the journey standing in a train corridor, the relics of two naval ratings traversed the hundred odd miles to London in the greater comfort of a carriage rack in a first-class compartment. The not unnatural desire to be relieved of the heavy burden when London was reached in the 'black-out' with an air-raid in progress and a total absence of transport was stoutly resisted, although the river under Waterloo Bridge was a sore temptation to a tired

Surgeon Rear-Admiral to shed his burden in the early hours of a winter morning. Those who know the charming story of San Michele will remember Axel Munthe's journey to Sweden so humorously told: this was my journey as a *Leichenbegleiter*—I am not anxious to resume the role.

In 1944 permission was granted by the War Office to the Medical Research Council for Dr. Joan Ross and Mr. Percy Hennell to visit the Central Mediterranean Front for the collection of suitable material and for the photography of what seemed appropriate to the needs of the Collection. The value of this step proved inestimable, and in Italy Lieut.-Colonel (now Professor) P. B. Ascroft, M.B.E., on his own initiative had also amassed a large congeries of injured brains, a selected number of which are now within the College.

Perhaps at this point a personal tribute may be offered to Dr. Joan Ross for her indefatigability as a collector as well as to her ability and learning as a pathologist. The Collection owes much to her effort and energy, and the Royal College of Surgeons of England demonstrated its appreciation of her work by awarding her in 1945 the John Hunter Medal and Triennial Prize for "her outstanding work in Pathological Anatomy, particularly in regard to the collection of pathological specimens illustrating injuries and diseases occasioned by war".

In this country throughout the War, and also for a period during the campaign in the Central Mediterranean, the valuable services of Mr. Percy Hennell for the taking of coloured photographs by a new process were made available by the magnanimous offer of the Metal Box Company Ltd. to the Medical Research Council. Thousands of specimens of this photographer's exquisite art in colour have been available for the Pathological Sub-Committee for its final selection, and four hundred colour photographs will shortly find a permanent place in the Royal College of Surgeons' Museum. These photographic records, whose range comprises such a motley group as wounds received in air raids, burns due to many agencies, dermatological features of disease, the muscle changes in crush injuries, the natural processes of repair, and the stages of operative interference, are faithful and exquisite representations of the originals. Some of the photographs have a historical interest and constitute mementoes of injuries incidental to the more novel methods of combat in the recent Armageddon, or reminders of a foul and degenerate nation's calculated inhumanity towards its prisoners of war. Such conditions as 'high-level' frost-bite due to exposure of the pilot to the slip-stream of a damaged aircraft, and the gangrenous feet of the unfortunate prisoners who tramped from Silesia to freedom come readily to mind.

Some of the illustrations from the pen and brush of Mrs. Gwen Trew were made on the battlefront in North-West Europe and include such a variegated collection as realistic representations of the ghastly damage inflicted on the lower extremity by the Schumine, the gangrenous processes in the distal limb that almost invariably follow injury to the popliteal vessels and only little less frequently to lesions of the femoral trunk, and the 'Kangaroo', the converted Canadian Ram Tank for bringing wounded from the battlefield.

Many other illustrations exhibiting this artist's skill have been made more recently in the tranquility of the Royal College of Surgeons or in the Middlesex Hospital from specimens contained in the War Collection. Considerations of expense and of space in this JOURNAL not unnaturally limit the amount of pictorial representation of the abundant material arranged around the Museum walls.

The Collection of specimens carefully selected which illustrate the results of the destructive agencies of modern warfare or constitute triumphant trophies of the surgeon's skill is now reaching its final number, and by the end of 1949 it is also expected that the 400 colour photographs which have been carefully chosen out of thousands as representative of many of the diseases and injuries of modern warfare and their repair by up-to-date methods will have reached their destination in the Royal College of Surgeons. It is hoped that the Army Authorities will permit the collection of pictures made by Mrs. Gwen Trew in the 'forward area' in the North-West Europe campaign to be handed over to the same body on permanent loan and for safe keeping.

My friends, Professor G. Hadfield and Dr. L. W. Proger, of the Royal College of Surgeons, have ever been ready to help me and there is one other to whom I would express my own thanks and the gratitude of those who study the Collection: W. J. Milton, in the service of the Medical Research Council, has been responsible for the mounting of the entire collection of specimens; no more conscientious and loyal worker could have been found for the responsible task.

Sir John Bland-Sutton, writing thirty years ago, refers to an interesting feature of the First War Collection that many of the communications which had appeared in medical journals during the actual years of the 1914–18 conflict and concerned gunshot injuries received in the War, relate to specimens contained in the Collection. Thus the First Great War Collection was not inaptly regarded as a Pathological Record Office. To a lesser degree the same is true of the more recent display. The present Collection will serve as a guide or signpost to the future war surgeon's problems, reminding him of the gaps in his knowledge, directing his reading, acquainting him with unfamiliar phenomena, stimulating his curiosity and whetting his zeal to equal and to endeavour to surpass the achievements of the veteran surgeons of a previous conflict.

INJURIES OF THE HEART AND BLOOD-VESSELS

A Harveian Lecture which concerns itself with the human jetsam of war should appropriately commence with the injuries and morbid conditions of that system of the human frame the mechanics of which the great physician did so much to clarify. The series of hearts in the First War Collection exhibiting the effects of gunshot injury was described by Sir John Bland-Sutton as "probably unique"; ten of these specimens mounted thirty years ago were figured in Sir George Makin's classical contribution in the *Official History of the Great War*. Although falling somewhat short of the First Collection in their abundance, the hearts in the recent series exhibit nearly all the features presented by

those placed in the Museum at the close of the 1914–18 conflict, but there is one novel cardiac lesion resulting from the calculated cruelty of a people that persistently violates every tenet of the Red Cross and surpasses in savagery the most ferocious beast—the Belsen heart, of which there are eight (NC.576, *Fig.* 175; 578; 580, *Fig.* 176; 612; 614; 615; 619; 623) in the Collection, constitutes a dual monument to the changeless sadistic brutality of the Nazi and to the long-drawn-out Gethsemane of his polyglot victims. Many of the specimens come from starved young women, usually suffering from advanced tuberculosis; the hearts are small, measuring from 7 to 7·8 or 8 cm. from base to apex, and weighing 110 to 190 g.; the heart shows brown atrophy: there is a marked diminution in the size of the great blood-vessels.

FIG. 175.—NC.576. Heart from woman dying in Belsen Concentration Camp. Note small size. (× ¾.)

These atrophic hearts themselves can never endure to become a *monumentum aere perennius* of callous murder, yet when they have crumbled and faded with the passage of the years the printed page and the illustrator's art will remind men of the hideous name of Belsen. *Caveant omnes*; the leopard does not change his spots. *Toujours le sale Boche*; Belsen—that is a name for ever to stigmatize an insensate, unrepentant people's calculated satanic ferocity.

Injuries of the heart had always fascinated Sir John Bland-Sutton, who, in his paper in the BRITISH JOURNAL OF SURGERY in 1919, may be said to have played the role of *compère* to the 1914–18 War Collection. I have in a previous paragraph envisaged this recent War Collection as perhaps functioning as a spur to self-searching for chinks in the armoury of our knowledge, and as a signpost to direct the visitor to the appropriate current and earlier literature. Those who wish to derive pleasure as well as information from their reading are referred to the addresses given by Bland-Sutton on the subject of cardiac injuries in 1910 and 1919 respectively. Death after an injury to the heart from a high-velocity bullet may be instantaneous, the dynamic disturbance causing it to stand still. If not immediately paralysed, the wounded heart usually bleeds so freely and rapidly that death follows in a few minutes. Yet in a group of cases where the heart has been struck by a fragment of high explosive of low velocity or a spent bullet, the victim may recover from the injury.

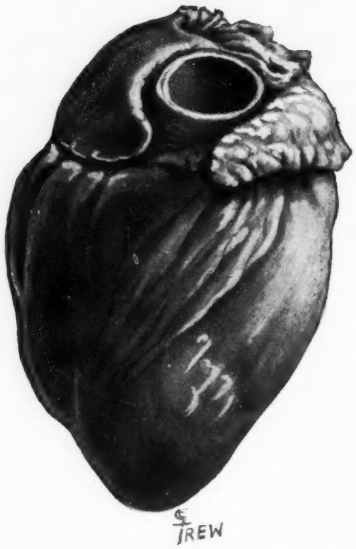


FIG. 176.—NC.580. Heart of girl of 17 dying in Belsen Concentration Camp. (× ¾.)

Every degree of severity of gunshot wound of the heart is to be found in the present Collection: the superficial laceration (CMF.61; CMF.289) from the fragment of high explosive *en passage*; the lodgement of the foreign body in the muscular wall of the heart, perhaps a minute piece of metal, as in the heart of the Greek sailor (CMF.149); perhaps a bullet in the wall of the left ventricle (NC.98) or one almost hidden under the posterior cusp of the mitral valve (NC.659, *Fig.* 177), having entered the left auricle between the pulmonary veins. Sometimes the missile has inflicted a through-and-through wound of a ventricle, small as in NC.166, from revolver ammunition, or so large as to be almost explosive in appearance (CMF.148, *Fig.* 178; NC.663). Occasionally the heart has been almost shot to pieces, as in NC.113 (Charles Warrick's case).

In his fascinating lecture on 'Missiles as Emboli' Bland-Sutton describes and figures several specimens where a fragment has been carried as an embolus and become arrested in the heart. The two earliest records of missiles entering the heart chambers as emboli which Bland-Sutton could find were those of Dr. Thomas Davis (1834) and of Fayrer and White (1854). In the former a piece of wood, after wounding the lung, had entered the vena cava and was carried via the right auricle to be arrested in the right ventricle; the second case was that of a soldier who died seventy-two days after wounding; the bullet apparently entered one of the left pulmonary veins and was arrested in the cavity of the

left ventricle near the apex. The 1914–18 Collection contained several specimens of fragments of metal arriving in the heart cavity as emboli: the recently acquired collection is not so rich in this type of specimen.

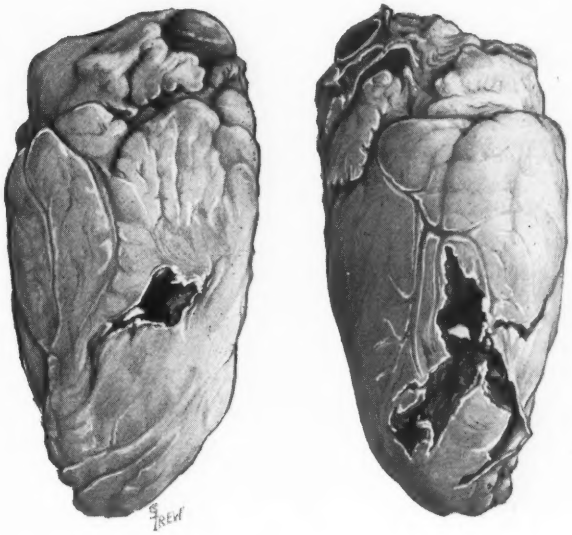
Specimen NC.385 (*Fig.* 179) came from a bombardier who sustained an abdomino-thoracic wound of the right side, the point of entry of the fragment being in the right 8th intercostal space. The man died suddenly twenty-four hours after a laparotomy, and at post mortem the missile was found to have traversed liver, diaphragm, and the antero-inferior aspect of the right auricle and to have lodged in a sinus of Valsalva at the root of the pulmonary artery.

The inclusion in the Collection of several hearts with a retained missile (NC.65; NC.98) not unnaturally raises the question of the propriety of the removal of these foreign bodies during life. This is no recent problem: during the last two conflicts the presence of a piece of metal in the

FIG. 177.—NC.659, showing bullet almost completely hidden under posterior cusp of mitral valve.

heart wall or within one of the cardiac chambers has provided an irresistible challenge to surgical courage and enterprise. The swirlings and gyrations of bullets and fragments of high explosive have been watched by radiologists and surgeons with the same interest that "astronomical observers bestow on minor planets", and these fascinating fluoroscopic séances have only served to whet the avidity of operators to rid the unfortunate owners of their unpleasant tenants.

Of the four cases of early removal of a metal fragment from the heart (the first to the fifteenth day after wounding) in the Casualty Clearing zone, recorded by Makins in his classical article in the *Official History of the Great War of* 1914–18, only one missile, removed by the late Sir John Fraser, Bt., seems to have been indubitably extracted from one of the heart chambers. Fraser's patient recovered, as did a man with a wound of the wall



A B

FIG. 178.—CMF.148. A, Wound of entry in right ventricle. B, Large triradiate exit wound on posterior aspect of left ventricle.

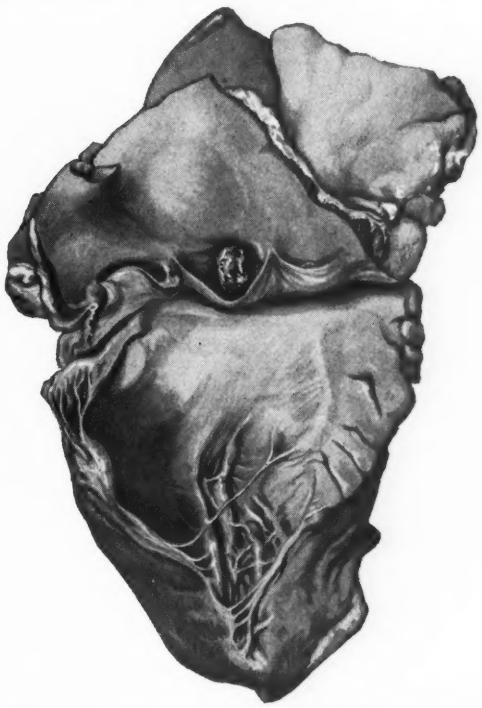


FIG. 179.—NC.385. Right-sided abdomino-thoracic wound; missile traversed liver, diaphragm, and right auricle and lodged in sinus of Valsalva at root of pulmonary artery.

of the left ventricle operated on by that distinguished surgeon, H. H. Sampson, of Birmingham. In two further cases a bullet was extracted from the muscular wall of the heart by the late Sir Henry Gray, of Aberdeen and Montreal, and by Walter Briggs, of Blackburn; these ended fatally, but in Briggs's case

operation was undertaken when severe pericarditis was already present.

During the First Great War some uncertainty prevailed as to the best method of removal of a foreign body, some French surgeons utilizing the method which Petit de la Villeon had successfully employed in the surgery of the lung, extracting metal fragments in the darkness of the X-ray room by a turn of bewildering necromancy.

During the last Armageddon, Dwight Harken, the Boston thoracic surgeon, operating at an American Chest Centre (160 General Hospital) in the South of England, successfully removed a fragment of metal from the heart cavities no fewer than thirteen times, although in the case of one patient the missile was only finally secured at the third intervention: there were no deaths in this remarkable series. This magnificent result fully justified a bold attitude to the problem of the foreign body in the heart. Surgery is indicated according to this Boston surgeon: (1) to prevent embolism of the foreign body or of an associated thrombus; (2) to reduce the risk of bacterial endocarditis, no fanciful danger. as demonstrated by the investigations on fragments surgically removed from the human heart and by animal experiment; (3) to avoid recurrent pericardial effusions; (4) to diminish the incidence of myocardial damage; and (5) pain and cardiac neurosis may become a compelling factor in arriving at a decision to operate.

On the other hand, since the war of 1914–18 many cases have come under observation in which missiles have been retained in the heart and the patients have been able to pursue more or less normal activities: for example, Grey Turner's patient was alive twenty-four years after with a bullet in the wall of his left ventricle. The Collection also includes one such heart (NC.98); the man, who had been wounded in the 1914–18 war, carried a bullet in the wall of his left ventricle till 1943.

Furthermore, the experimental work of Fritz, Newman, Jampolis, and Adams (1949) showed that, so long as foreign bodies retained in or about the heart are not producing symptoms or signs of cardiac dysfunction " they can be allowed to remain ". These observers found that foreign bodies placed in the myocardium do not tend to migrate into a chamber of the heart, and practically all are embedded in fibrous tissue in eight weeks. However, there can be no question as to the very real proclivity of foreign bodies within the cardiac cavities to embolize, and the authors found that only 3 out of 35 foreign bodies introduced directly into the chambers of the heart remained within the heart; these had apparently been entangled between the chordæ tendineæ, producing no untoward symptoms. Harken's* work has been emulated by Miscal, his colleague from the United States, who removed 39 foreign bodies " in the heart " without a death. D'Abreu, of Birmingham, successfully removed five fragments from the heart.

The paucity of opportunity enjoyed by British surgeons for the removal of fragments from within the heart cavities compared with their American colleagues might be taken to suggest a tougher ventricular muscle in their British Allies (!), but the numbers concerned are too small to be taken seriously.

Despite the brilliant achievements of these young surgeons I still remain lost in wonderment and admiration for the dramatic extraction of a bullet from the intrapericardiac portion of the inferior vena cava in 1918 by that great French surgeon, Pierre Duval, who did so much to promote the operative treatment of gunshot wounds of the thorax in the First Great War. The story was dramatically told: the fluoroscopic appearance of the bullet rising and falling above and below the diaphragm in the vena cava; the carefully devised median thoracolaparotomy; the thrill experienced in feeling the bullet in mid-stream slipping between and past his fingers four times, and even when he did manage to seize the missile, it again escaped twice from his grasp. The bullet was finally caught at the junction of the right auricle and vena cava. A 'purse-string' was inserted; an incision in the vein, bullet extracted, the thread drawn tight—" un petit jet de sang au niveau d'un des points: une pince, une ligature laterale. La suture est parfaitement étanché."

Some of the injuries to the heart and the larger blood-vessels in its immediate proximity produced

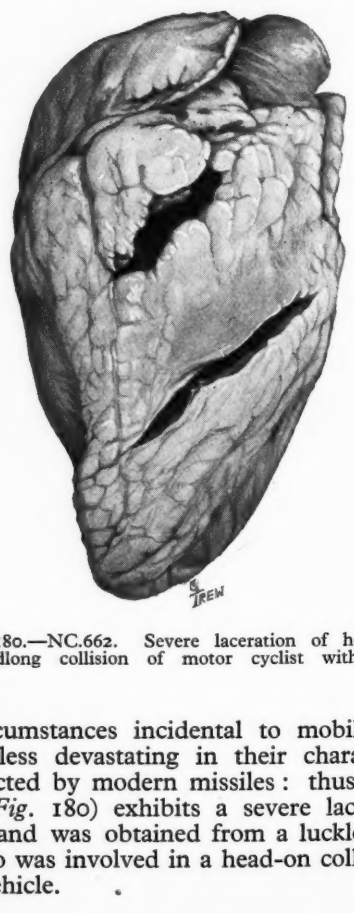


FIG. 180.—NC.662. Severe laceration of heart from fatal headlong collision of motor cyclist with another vehicle.

under circumstances incidental to mobile warfare are little less devastating in their character than those inflicted by modern missiles: thus specimen NC.662 (*Fig.* 180) exhibits a severe laceration of the heart and was obtained from a luckless motor-cyclist who was involved in a head-on collision with another vehicle.

* Harken removed 134 missiles from the mediastinum without a death, including 55 fragments on or within the heart (13 from the chambers of the heart), and 3 from the lumen of large blood-vessels.

Again, specimen CMF.36, from a man who was knocked down by a motor wagon, shows a rupture of the left ventricle which has almost an explosive character. An extraordinary injury was sustained by an officer who was thrown out of a vehicle in which he was travelling and killed. At the autopsy performed at the 15th Scottish Hospital in Cairo the sternum, ribs, and spine were found to be intact; the left pleural cavity contained 3 pints of blood, and just where the aortic arch curves over the left bronchus and 1 in. distal to the origin of the left subclavian artery *the aorta was split transversely* for seven-eighths of its circumference, the intact strip being uppermost (NC.525). The heart and pericardium showed no sign of any injury.

The few specimens of traumatic rupture of the heart not caused by missile injury which find a place in the Collection yield no reliable information as to the proclivity of this or that cardiac chamber to this lesion. Curiosity may profit from the perusal of the paper of Bright and Beck (1934), who found little difference in the liability of the cardiac cavities to suffer rupture, and in 8·5 per cent more than one cavity was involved. Those who are desirous of further information on the specific causation and the clinical features presented by the lesion are referred to Bright and Beck's communication.

The subpericardial hæmorrhage seen in specimen CMF.84, which resulted from blast from an exploding bomb, is similar to that encountered in certain cases of contusion reported by W. E. Errington Hume in his Medical Society Oration for 1941, in which the distinguished Newcastle physician includes a historical case described by Mark Akenside, the physician-poet, in 1763, where death ensued six months after a contusion of the chest and "a livid spot the size of half-a-crown was found post-mortem, bruised and jelly-like, near the apex of the left ventricle".

The injuries to the heart produced by the cult of speed vary in degree: one heart (CMF.416) merely showed extensive subendocardiac hæmorrhages on the left side of the interventricular septum. Its owner was rendered unconscious when his motor-cycle hit a post, the rider turning over and over several times.

Comparable subendocardial hæmorrhages and also in the heart muscle, especially that of the right ventricle, have been produced experimentally (Kulbs and Strauss, 1909; Schlomka, 1940; Bright and Beck, 1934) and such symptoms and signs ensued as bradycardia and extrasystoles, and perhaps later ventricular fibrillation and death; in some animals radiological enlargement was produced, persisting for a couple of weeks. These authors found, however, that the fatal results in the experiment were often out of all proportion to the post-mortem findings.

It is always difficult in the human being to be certain of the degree of injury to the heart when the patient recovers from a cardiac contusion; doubt may even be experienced whether there has been any cardiac damage at all. Such clinical phenomena as precordial pain, a 'knock out' from the injury, faintness, cyanosis, sweating, cardiac irregularity, tachycardia, orthopnœa, and perhaps electrocardiographic changes, either singly or in combination, are suggestive of cardiac contusion.

Other morbid conditions of the heart in our recently acquired collection include such a miscellany as subpericardial hæmorrhages scattered over the surface of the heart from a case of fatal anterior poliomyelitis (CMF.281), an infarct of the heart following sepsis of the pelvis and hip-joint (CMF. 259), a heart from a fatal case of heat stroke (CMF. 239), where there are to be seen a few minute hæmorrhages under the pericardium along the course

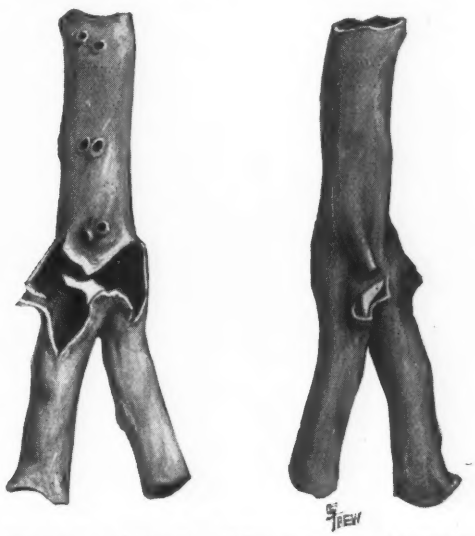


FIG. 181.—CMF.154. Bullet wound of bifurcation of aorta, the man surviving one-and-a-half hours. Note small entry wound on the anterior wall, and the much more extensive exit wound posteriorly. (*Presented by Major Roland Thornley, F.R.C.S.*)

of the right coronary artery, and along the course of the interventricular branch of the left coronary artery. Wilson in 1940 had drawn attention to the autopsy finding of a fairly extensive hæmorrhage of the ventricular septum in the vicinity of the bundle of His: a similar lesion is to be found in this specimen also.

The specimens exhibiting cardiac injuries and lesions due to the circumstances of war are not an insignificant group; there is, however, one type of rupture not exemplified in the War Collection: coronary occlusion and its sequelæ are not maladies of the combat years of a warrior's life. In a Harveian Lecture it is not inappropriate to quote a case of non-traumatic rupture of the heart reported by the great physician whose name this Society perpetuates. Harvey, in the presence of his friend, Dr. Argent, and Dr. Gorge, the Minister of the Parish, performed a post-mortem examination on Sir Robert Darcie, who in life had apparently suffered from angina pectoris and "at last opprest in a single paroxism, he dyed". They found a hole in the left ventricle which admitted easily one of Harvey's fingers, "from which blood had poured forth".

Commonsense suggests the gravity of wounds of the aorta: their severity is attested by the various charts in Cuthbert Wallace's *War Surgery of the Abdomen* (1918) indicating the wounds of entry and also the tracks of projectiles, which demonstrate the comparative absence of wounds in the midline in those with abdominal wounds reaching casualty clearing hospitals; men shot in this situation usually

die on the field of battle. Naturally the size, nature, and velocity of the missile are of importance in determining the type of injury inflicted. The curious will find the story of those who have survived injury to the aorta for days, months, or years in the works of such authors as Makins (1919, 1922) or Bland-Sutton (1920, a, b).

In the present Collection there is a specimen, CMF.154 (*Fig.* 181), of a wound of the abdominal aorta where a Naval rating, hit by an Italian

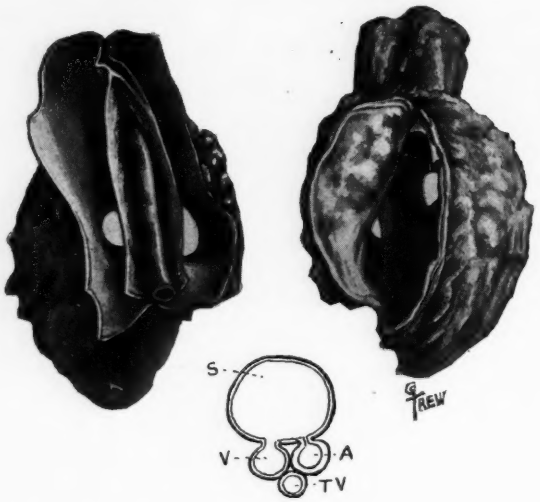


FIG. 182.—N.C.425. Traumatic arteriovenous aneurysm of femoral vessels in mid-thigh. Excision more than four years after wounding. Artery and vein communicate with a common sac containing laminated clot. Between the artery and vein is a venous tributary joining the femoral vein at its upper end. A. Artery; V, Vein; S, Aneurysmal sac; TV, Thrombosed vein. (*Sir James Paterson Ross, K.C.V.O., F.R.C.S.*)

bullet in the streets of Naples, survived one and a half hours. The wound of exit on the posterior wall of the aorta was much larger than the wound of entry; the whole of the retroperitoneal tissue was infiltrated with a massive effusion of many pints of blood extending vertically from the diaphragm to the pelvis, and from ascending to descending colon in the transverse direction. It seems scarcely possible that the man could have continued to live for ninety minutes with his aorta wide open.

Another specimen of rupture of the aorta as well as the auricle is seen in NC.158, which was obtained from a lorry driver who was killed outright through a 'head-on' collision with a telegraph post in the 'black-out' (presented by Dr. E. Biddle).

On the other hand there are in the Collection two cases of injury to the oarta that survived about six months. A man received multiple wounds of the back from a mine explosion and died 186 days after wounding; a traumatic aneurysm of the abdominal aorta had ruptured into the left pleural cavity producing extreme mediastinal displacement with compression of the left lung (NC.91) (Lieut.-Col. J. H. Gear's case). Another specimen is that of a traumatic aneurysm of the abdominal aorta produced by a fragment of high explosive, the patient surviving more than six months. NC.383 shows an *incomplete tear* of the thoracic aorta in the posterior mediastinum of a left-sided abdomino-thoracic air-raid casualty, who had also suffered an injury to lung, diaphragm, and spleen.

There is also a specimen (NC.108) showing a hole in the base of the *pulmonary artery*: the pericardium was full of blood; multiple injuries were produced by the explosion of a box of detonators.

The mantle of Sir George Makins, whose experience of gunshot wounds of the blood-vessels was remarkable and whose judicial pronouncements on the problems raised by the sequelæ of arterial injury have always commanded the greatest attention, has descended on his former pupil and colleague, Bernard Maybury, and his contributions to the *Bulletin of War Medicine* are recommended to the notice of those interested in the subject of traumatic aneurysm. There are specimens of traumatic aneurysms in the Collection, but the arteriovenous fistula and aneurysm is perhaps always to be regarded as the special feature, the *spolia propria*, of any War Museum. According to Maybury, operation for traumatic false aneurysm may generally be deferred for three or four months, although circumstances may compel earlier interference: surgical treatment for varicose aneurysm should not be undertaken for four to six months, and that for aneurysmal varix for six to twelve months or even longer. Arteriovenous (varicose) aneurysms lend themselves readily to excision: post-operative anxieties are less; these specimens are therefore fairly numerous on our shelves; amongst them are NC.425 (*Fig.* 182—Sir James Paterson Ross), NC.216 (Charles Donald), CMF.117 (P. B. Ascroft), and CMF.439 (C. Parish).

Some of the traumatic aneurysms involving the brachial artery have been ablated by surgeons such as E. A. Jack (NC.189), D. L. Griffiths (NC.418), P. B. Ascroft (CMF.117, *Fig.* 183), and W. R.

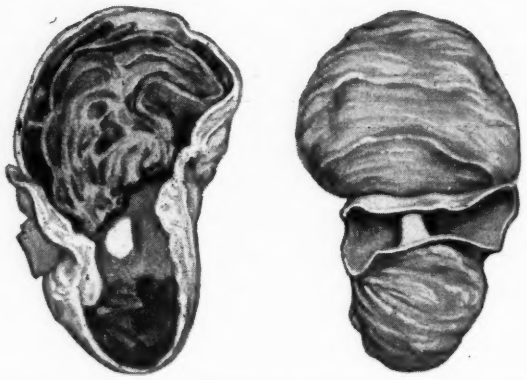


FIG. 183.—CMF.117. Traumatic aneurysm of brachial artery. Excision. (*Lt.-Col. P. B. Ascroft, M.B.E., F.R.C.S.*)

Merrington (CMF.377, *Fig.* 184). There are traumatic aneurysms of the carotid—NC.237 (Michael Oldfield), NC.216 (Charles Donald), NC. 198 (P. B. Ascroft), CMF.120 (P. B. Ascroft, *Fig.* 185), and NC.94 (R. S. Handley); the vertebral, involving ligature of the first part of the subclavian and excision of the sac—CMF.116 (Ascroft), and CMF.119 (Ascroft); an axillary aneurysm, CMF.118 (Ascroft) also finds inclusion, and several radial arterial aneurysms, which are usually 'easy money', as well as an aneurysm of the superficial temporal artery, CMF.28 (Guy Blackburn, M.B.E.).

An interesting specimen (CMF.120) is that of a 'berry' aneurysm on the wall of the common carotid (*Fig.* 185) resulting from a through-and-through wound of the neck, which ended fatally from sepsis arising from other wounds (P. B. Ascroft).

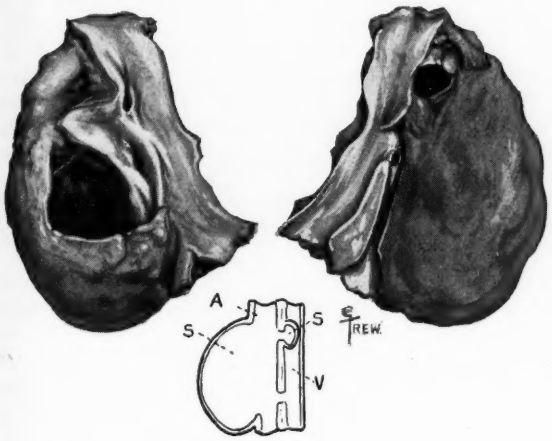


FIG. 184.—CMF.377. Arteriovenous aneurysm of brachial artery. With diagram indicating disposition of sacs and communications. Excision 36 days after wounding. A, Artery; V, Vein; SS, Aneurysmal sacs. (*Major W. R. Merrington, F.R.C.S.*)

In Paterson Ross's patient (NC.425, *Fig.* 182) the arterial injury was not recognized till five months after the injury, but it was four years after wounding by an aerial bomb fragment before a successful excision of the arteriorvenous aneurysm of the femoral in Hunter's canal was performed.

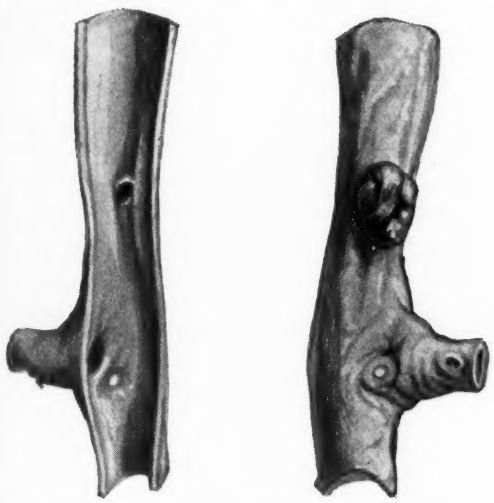


FIG. 185.—CMF.120. 'Berry' aneurysm of common carotid resulting from a through-and-through wound of the neck. (*Professor P. B. Ascroft, M.B.E., F.R.C.S.*)

Over forty years had elapsed since Ungley's patient had received a gunshot wound in the Boer War traversing the upper part of both thighs from left to right, the wound of exit being on the lateral aspect of the upper right thigh: the arteriovenous aneurysm involved the right common femoral artery and vein at the origin of the profunda (NC.83, *Fig.* 186). A quarter of a century elapsed before he suffered much disability from the abnormal communication, but for fifteen years he had had intermittent ulceration at the ankle and latterly pain brought him to operation (Gordon Ungley's case).

Traumatic dissecting aneurysm of the left internal carotid artery. Specimen CMF.230 (*Fig.* 187) was

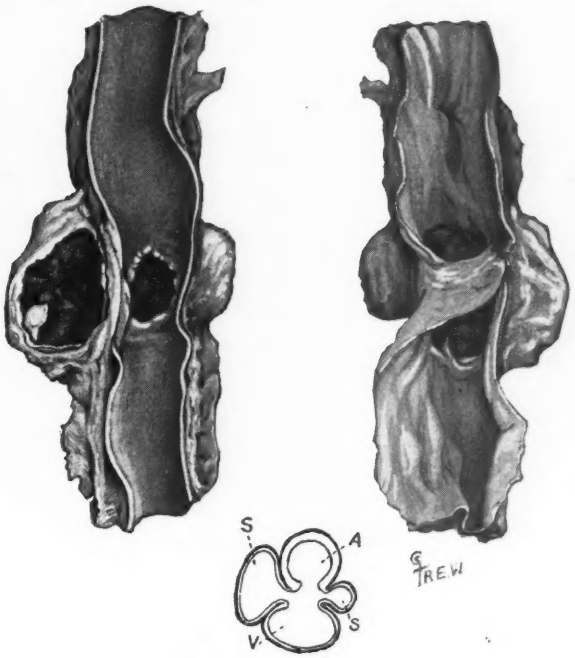


FIG. 186.—N.C.83. Arteriovenous aneurysm of common femoral artery resulting from bullet wound in Boer War. Operation on account of pain and ulcer of leg 45 years after. A, Artery; V, Vein; SS, Aneurysmal sacs. (*Gordon Ungley's case.*)

FIG. 187.—N.C.230. Traumatic dissecting aneurysm of left internal carotid artery. Man knocked down from behind by rope attached to a lorry. Rope wound itself around his neck, threw him to the ground, then unwound without dragging him along. The original lesion had been a hæmorrhage into the media of the arterial wall and the formation of a dissecting aneurysm with subsequent thrombosis. The stripped-up intima and two-thirds of media blocks the arterial lumen. (*Major A. D. Morgan.*)

obtained from a man who was knocked down from behind by a rope attached to a passing lorry: the rope wound itself round his neck, threw him to the ground and then unwound itself without dragging him along. A complete right-sided hemiplegia developed twenty-four hours after the accident and death ensued twenty-four hours later. *Post-mortem*: a distinct pressure mark could be seen round the

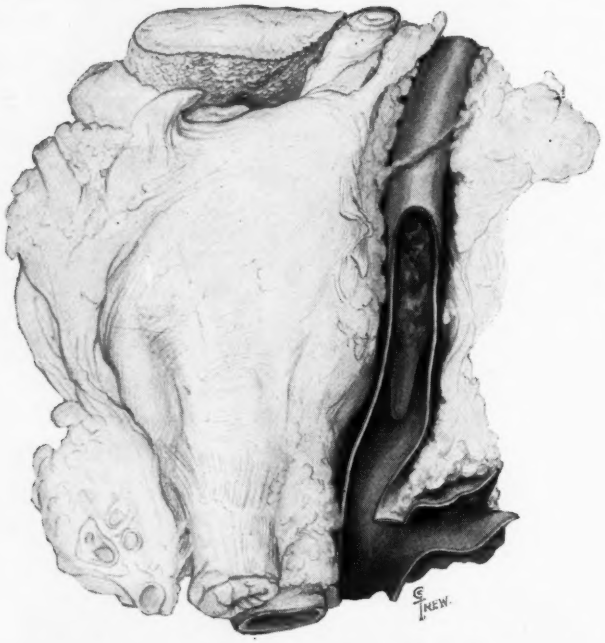


FIG. 188.—CMF.273.—Through-and-through wound of neck producing a contusion of internal carotid, with immediate thrombosis. Viewed from behind.

neck 2½ in. below the right ear. The first inch of the left internal carotid artery was transformed into a hard bluish spindle-shaped swelling as a result of thrombosis: the thrombus extended into the left anterior and middle cerebral vessels, resulting in a softening of the left cerebral hemisphere.

The original lesion had been a hæmorrhage into the media of the arterial wall at the seat of the injury and the formation of a dissecting aneurysm with subsequent thrombosis. The specimen shows the stripped up intima and two-thirds of the media blocking the arterial lumen (*Fig.* 187). The right artery has a patch of atheroma at the corresponding level (Major A. D. Morgan).

Contusion of an artery with thrombosis and occlusion of the lumen of the vessel as the result of the passage of a missile by a track coursing in close proximity to the artery has been a phenomenon familiar to military surgeons of several generations. *Fig.* 188 (CMF.273) shows a contusion of the internal carotid with immediate thrombosis resulting from a through-and-through wound of the neck behind the pharynx.

ABDOMINAL INJURIES

These were not without interest to John Hunter, and in the old Collection was a specimen of a gunshot wound of the small intestine (*Fig.* 189) resulting from a duel in Hyde Park. Many of the mounted specimens in the Collection which illustrate the abdominal injuries of warfare have already been described and figured in communications by the writer during the war years, and the reader is referred to the author's Bradshaw Lecture (1942), a Hunterian Lecture (1944), and a paper entitled 'The Last Hate', all of which were published in the BRITISH JOURNAL OF SURGERY; but the Collection also includes other specimens which have not been figured in the various communications referred to. Among these are several specimens representing the effects of blast on the abdominal viscera: specimen NC.78 (Sir Cecil Wakeley, *Fig.* 190) demonstrates the intestinal lesions produced by experimental 'immersion blast': another specimen (NC.537) shows the effects of blast on the small intestine of a soldier from the close explosion of a mortar bomb: a large rent involving half the circumference of the bowel and an inch of mesentery necessitated the resection of 6 in. of ileum: the man recovered (F. D. Hindmarsh): while yet another (CMF.57, *Fig.* 191) shows a blast injury of the small gut, which is purplish in colour, with

FIG. 189.—*Hunterian specimen.* Small intestine from a man fatally wounded in a duel in Hyde Park. (*From a photograph in the writer's possession.*)

two linear tears through the serous coat: the lumen of the bowel contains much altered blood (Lt.-Col. G. J. Cunningham, M.B.E.).

Several of Percy Hennell's coloured photographs concern the treatment of injuries of the abdominal viscera. *Fig.* 192 depicts an exteriorized right-sided colon: the ileocæcal orifice is also presenting in the everted mucosal surface of the cæcum: the excoriation of the skin, which is often so distressing in

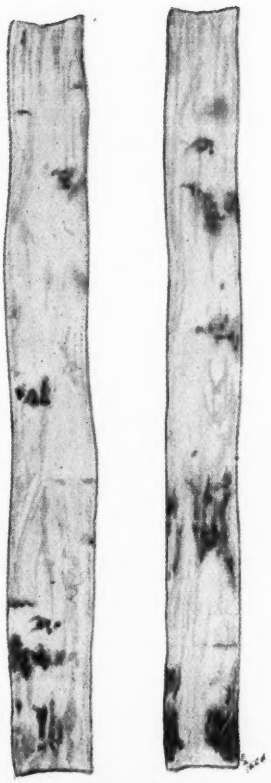


FIG. 190.—NC.78. Goat intestine, showing changes produced by exposure to 'immersion blast'. (*Sir Cecil Wakeley, K.B.E., C.B., F.R.C.S.*)

FIG. 191.—CMF.57. Blast injury of small intestine. Ten feet of small gut, dark purple in colour: lumen of bowel contained much altered blood. One or two linear tears through serous coat of stomach and small gut. (*Presented by Lt.-Col. G. T. Cunningham. M.B.E.*)

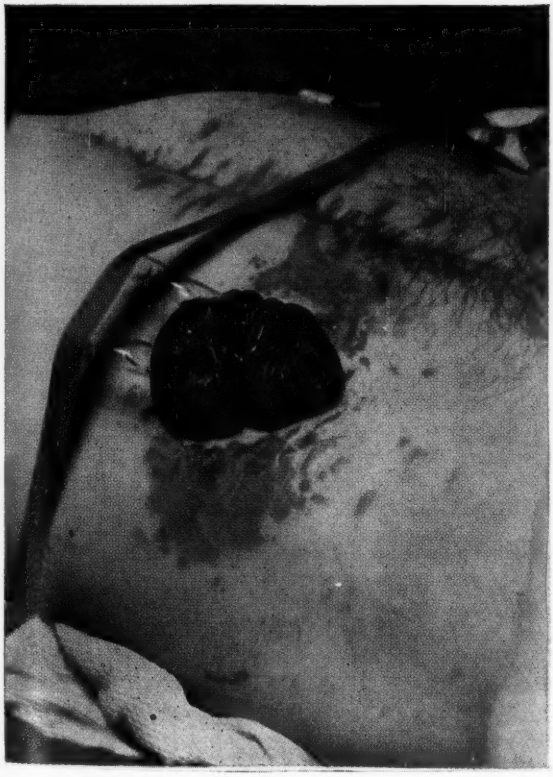


FIG. 192.—Gunshot wound of abdomen: exteriorized right-sided colon: the excoriation of the skin which is often so distressing a feature of right-sided colon exteriorizations is well shown. (*Photograph by Percy Hennell.*)

FIG. 193.—One of Percy Hennell's coloured photographs showing 'defunctioning' transverse colostomy in a naval surgeon with shattered iliac colon. (*Sir Cecil Wakeley, K.B.E., C.B., F.R.C.S.*)

right-sided exteriorizations of the large bowel, is well shown. Another illustration (*Fig.* 193) reproduces the abdominal wall of a patient under the care of the President of the Royal College of Surgeons, Sir Cecil Wakeley, who had suffered a severe wound of the left-sided colon and who, in order to ensure a successful result, had been submitted to a 'defunctioning' transverse colostomy before the repair of the shattered distal colonic segment was undertaken.

A volvulus of the small intestine initiated by torsion of a 5-in. Meckel's diverticulum on its own axis (CMF.24, *Fig.* 194) necessitating a resection of a foot or two of ileum in a malarial subject, made diagnosis difficult because of the possibility

FIG. 194.—CMF.24. Torsion of a Meckel's diverticulum initiating torsion of the ileum in a soldier with malaria. Successful resection of bowel. (*Rodney Smith, F.R.C.S.*)

of the colic being a toxic phenomenon due to the administration of pamaquin, or from the simulation of an acute surgical catastrophe by intestinal capillary thrombosis such as may occur in malaria. The emergency arose on the very morning of the commencement of the battle of the Sangro: the man was fortunate, since it was safer to be in the hands

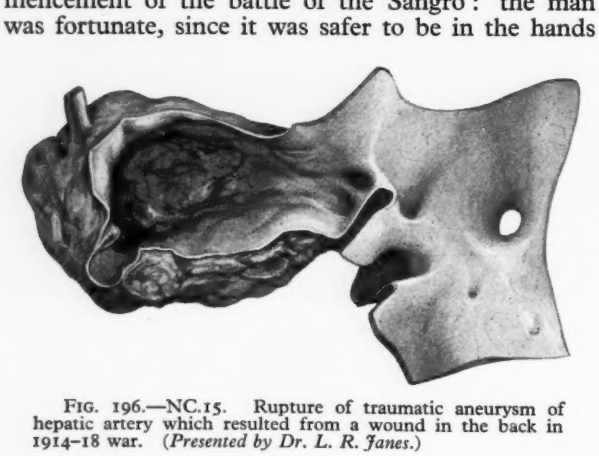


FIG. 196.—NC.15. Rupture of traumatic aneurysm of hepatic artery which resulted from a wound in the back in 1914–18 war. (*Presented by Dr. L. R. Janes.*)

of Rodney Smith than to share the fortune and fate of his comrades in battle (!).

In a war waged on many continents and on the Seven Seas considerations of transportability must have weighed with those whose concern was with amassing a central assemblage of pathological material. Despite the eagerness of many to facilitate the homeward conveyance of surgical and pathological trophies, there were naturally more important and cogent priorities: the number of square inches and of pounds avoirdupois played a part in unlocking the hatches and doors of ships and aircraft. There is, therefore, not the same luxuriant profusion of specimens demonstrating *missile injury to the liver* as the first Collection could boast (*Fig.* 195), although

FIG. 195.—CMF.329. Liver showing track of bullet from 'Tommy' gun: thoracico-abdominal wound. Death two days after injury. (*Major H. W. Burge, M.B.E., F.R.C.S.*)

there are several which were retrieved from unfortunates killed by modern Juggernauts on the roads of Great Britain and elsewhere. The 'mailed fist' of the radiologist has been known to rupture a spleen, to perforate a peptic ulcer, or determine a hæmatemesis or melæna (!), but NC.15 (*Fig.* 196) is a *ruptured traumatic aneurysm of the hepatic artery* consequent on a wound in the back during the 1914–18 War. During the necessary manipulation of the abdomen associated with a barium-meal investigation the aneurysm ruptured into the peritoneal cavity: a fragment of high explosive was found embedded in scar tissue under the left cupola of the diaphragm (L. R. Janes's case).

Specimen CMF.375 is an *aneurysm of a branch of the hepatic artery* producing fatal hæmorrhage in a man who had received a right-sided abdomino-thoracic bullet wound. The hæmorrhage came from a small aneurysm projecting from the wall of an abscess cavity in the liver (Major C. J. Cunningham). By contrast, however, with more massive solid viscera the less obtrusive *gall-bladder* finds a home in the Museum: NC.783 (*Fig.* 197), exhibiting a through-and-through wound, is an almost exact replica of a specimen in the 1914–18 Collection (W. O. Coll. 1042A), whose place it may be regarded

as having taken. Among other things curious and small is a specimen (NC.26, *Fig.* 198) presented by A. H. T. Robb-Smith, which shows a *contusion of the suprarenal* following a lorry accident.

Doubtless the plethora of *spleens* in the Collection finds its explanation in facility of transport as well as in the ease wherewith the organ may be removed when a transdiaphragmatic approach is employed, a route which, though very familiar to operators of a former campaign, not unnaturally

FIG. 197.—NC.783. Thoraco-abdominal wound, traversing gall-bladder.

intrigued the more youthful generation of war surgeons, and which, " since the bugles sang truce ", has been exploited by them to advantage in thoracico-abdominal gastrectomy for cancer of the upper segment of the stomach. None of these spleens can boast the same dramatic story as that removed by Richard Charles of Ipswich in the First Great War, which found a place in the old War Museum. The owner of that spleen was the possessor of the Victoria Cross, commanded his Canadian regiment and led his troops across the Rhine over the Hohenzollern Bridge at Cologne on the occasion of the Armistice in 1918, subsequently returning to this side of the Atlantic a Major-General in the Canadian Forces during the last great conflict.

In view of the regions of the globe over which the late war was fought, more ruptured malarial spleens might have been expected: one such specimen (NC.505) was successfully removed from a soldier who injured his spleen by unluckily falling over a tent-rope on to a tent peg (!): the spleen weighed 630 g. and measured $6\frac{1}{4} \times 4\frac{1}{4} \times 1\frac{3}{4}$ in. (Michael Oldfield).

The *kidneys* in the Collection rival the spleens in numbers (*Fig.* 199); they, too, are of course easy of transport, but their abundance also bespeaks the realization of observers how frequently ' traumatic uræmia ' ends the scene in those who are casualties of war. Unfortunately such specimens do not make good museum pieces, and the clinical history and the microscope are required to allocate the specimen to its proper category, e.g., transfusion kidney, crush kidney, the fat embolism kidney, etc.

FIG. 198.—NC.26. Traumatic hæmorrhage of suprarenal—man knocked down by an army lorry. Other severe injuries. Death in 12 hours. (*Presented by A. H. T. Robb-Smith, Radcliffe Infirmary, Oxford.*)

Some of the more grossly damaged kidneys, e.g., those due to enemy missiles, have already been figured in papers by the writer—for example, a kidney damaged by a fragment of a V1 bomb (NC.397) (John Everidge's successful case).

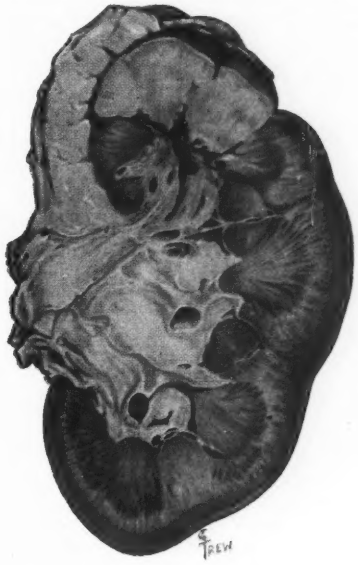


FIG. 199.—CMF.389. Kidney: Thoraco-abdominal bullet wound necessitating splenectomy. Death two months later; bullet had passed through upper pole of kidney, cutting off blood-supply, so that it has the appearance of an old white infarct.

The ' Belsen kidney ', accursed name, presents nothing worthy of comment except its small dimensions (NC.614; NC.615).

Chiselled in stone above the entrance to the old post-mortem room at Middlesex Hospital, but now

emblazoned in gold letters above the more recent one, are the words: *Hic locus est qua mors gaudet succurrere vitæ.* That sentence well describes the lesson to be learned from the specimens of *pulmonary concussion* ('blast lung') in the Collection (NC.7, *Fig.* 200; NC.8), which, as Professor

FIG. 200.—NC.7. Blast injury of the lung; large part of lung consolidated. Dark consolidated areas had ill-defined margins which were bright red, fading off into lung of normal colour. Small ashen-grey foci scattered throughout dark consolidated lung. (*Presented by Professor G. Hadfield.*)

Hadfield pointed out, demonstrated the progressive nature of the pulmonary hæmorrhages and indicated that one of the chief principles of treatment must be to immobilize the patient as soon as possible, and inflict on him a minimum of disturbance for a period of several days. The second specimen of 'blast lung' (NC.8) also retains the colour associated with carbon-monoxide poisoning, and the lesson to be learned was the added danger to life resulting from bursting gas mains from 'incidents' connected with aerial attack. NC.806 is the contused lung of a child 1 year old who was hurled against the wall of a room by blast.

HEAD INJURIES

Juvenal's *insanabile scribendi cacoethes* may be no inaccurate description of Britain of to-day with its hecatombs of paper sacrificed to the mechanical recording by dexterous, if time-conscious, fingers of the ullage of dull people's brains, but an *insanabile colligendi cacoethes* or enthusiastic acquisitiveness most certainly characterized the activities of Ascroft, who amassed and transported to this country over 200 brains for use in the study of cranial injuries. In the special War Surgery Supplement of the BRITISH JOURNAL OF SURGERY devoted to Head Injuries the pages of Ascroft's communication entitled "A Survey of Fatal Head Wounds" are interspersed with illustrations depicting the appearance of some of the specimens now in our Collection, but specimens CMF.517 (*Fig.* 201), CMF.518 (*Fig.* 202), CMF.521 (*Fig.* 203), and CMF.400 (*Fig.* 204), are not figured in the article referred to and are reproduced here.

FIG. 201.—CMF.517. Coronal section through cerebellum showing hæmorrhages into pons and fourth ventricle. Blunt injury: immediate death. (*Professor P. B. Ascroft, M.B.E., F.R.C.S.*)

FIG. 202.—CMF.518. Shows effect of blunt injury to parieto-occipital cortex: cortex hæmorrhagic and destroyed. (*Professor P. B. Ascroft, M.B.E., F.R.C.S.*)

Specimen CMF.517 (presented by P. B. Ascroft) is a coronal section through the cerebellum showing hæmorrhages into the pons and fourth ventricle; the man had suffered a blunt injury to the head and was admitted to hospital dead; fissures of the vault of the skull radiated to the base; apart from the lesions depicted here there were diffuse hæmorrhages in the cerebral cortex. Specimen CMF.518 (Ascroft) is a coronal section through the posterior part of the parietal lobes of the brain, showing the

effects of blunt head injury. Brain area 3 × 3 cm. of the left parieto-occipital cortex is hæmorrhagic and destroyed. Specimen CMF.521 (Ascroft) is a coronal section through the temporo-parietal region of the brain, showing the through-and-through track of a revolver bullet. The patient survived twenty-eight hours; the track contained blood, debris, and small loose bone-chips.

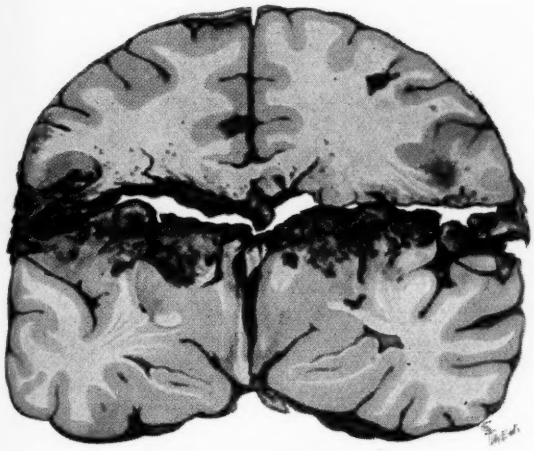


FIG. 203.—CMF.521. Through-and-through track of revolver bullet across temporo-parietal region of brain. (*Professor P. B. Ascroft, M.B.E., F.R.C.S.*)

A more subtle pathological cause than blunt trauma to the head is responsible for the beautiful specimen NC.29 (*Fig.* 205) of a brain exhibiting the 'cayenne pepper' appearance all through the white matter which is so characteristic of fat embolism. There is, however, no poverty of specimens of cerebral fat embolism: the pin-point petechial hæmorrhages have a special predilection for the neighbourhood of the central nuclei of the cerebral hemispheres. The protracted war taught observers to be cognizant of the necessity of giving consideration to the possibility of fat embolism, a blast injury, or the crush syndrome in patients labelled 'surgical shock'.

A debt is owing to Sir Cecil Wakeley and Sir Alun Rowlands and Dr. A. H. T. Robb-Smith for early drawing the attention of war surgeons to the condition, and the considerable bibliography cannot but be useful for those whom it behoves to acquaint themselves with the subject.

MISCELLANEOUS

One of the remarkable features of the present Collection is the paucity of specimens of damaged bone, thereby constituting a tribute to the work of the Forward surgeon, wonderful transport, and the amazing properties of penicillin.

When it is remembered that in the later years of the recent war only 9 *per cent* of the compound fractures treated by delayed primary suture and only 7 per cent of those dealt with by secondary suture in the Italian campaign showed *failure of healing*, an explanation is forthcoming for the few specimens displayed of macerated bones exhibiting projectile fractures. The contrast in the matter of numbers of specimens demonstrating bone pathology from the 1914–18 War is, at any rate, most marked, when the fracture material collected at the disposal of museum workers led to the publication of three beautifully-illustrated papers by Sir Arthur Keith and Captain Morton E. Hall, C.A.M.C., in the pages of the BRITISH JOURNAL OF SURGERY. The Hunterian Lecture of the present Chairman of the Editorial Committee of the BRITISH JOURNAL OF SURGERY, E. K. Martin, on 'Projectile Fracture of Limb Bones', in which he stressed the role of bruising

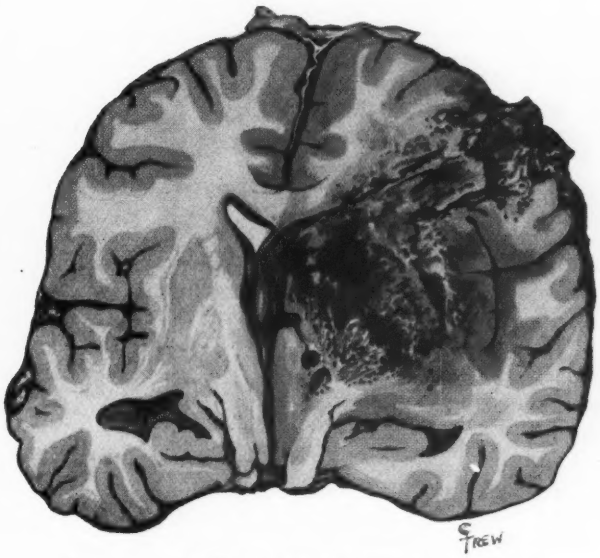


FIG. 204.—C.M.F.400, showing extensive damage to cortex and basal nuclei.

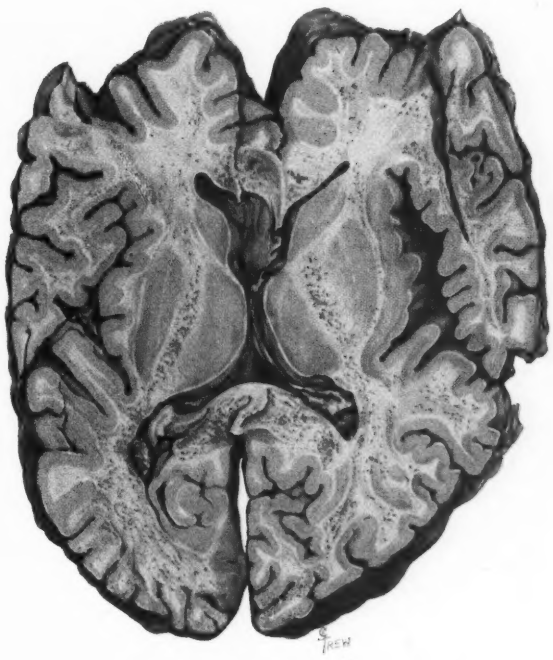


FIG. 205.—NC.29. Showing the 'cayenne-pepper' appearance in the white matter of the cerebrum, so characteristic of fat embolism.

FIG. 206.—Two typical cases of traumatic amputation by Schumine.

FIG. 208.—CMF.264. Gangrene of foot following ligature of popliteal vessels; fascial decompression 'slash' in calf of leg performed at same time. Amputation. Pallor and ischæmia of muscles, especially in anterior compartment of leg and also soleus. Widespread thrombosis of blood-vessels. (*R. J. Furlong, F.R.C.S.*)

of the cancellous tissue and fissuring of bone in the spread of infection, was based on no inconsiderable amount of pathological material.

Such mounted specimens of the limbs as are to be handled on the shelves or found among the series of coloured photographs and other illustrations are the traumatic amputations and the devastating effects of the Schumine and other examples of ingravescent human devilry in the art of machination (*Fig.* 206).

FIG. 207.—NC.3. Crush injury of muscle. Air-raid casualty: buried 3 hours. The specimen shows widespread interstitial hæmorrhages: an ill-defined boundary zone of very pale muscle is visible in some areas. Note the pallor and opaque appearance of muscle-fibres. (*A. H. Hunt, F.R.C.S.*)

So frightful are the effects of these mine injuries that some effort may be even required of the visitor to discern and decipher the origin of the mangled and disrupted human part displayed.

The lesions of the soft tissues seem to have been more attractive to collectors in the late conflict, and there are specimens of crush injury to muscle (NC.3, *Fig.* 207), and of ischæmia of muscles of the leg (CMF.264, *Fig.* 208).

Illustration 899 (*Fig.* 209) reproduces the typical reaction to liquid mustard on damp areas of the body. The patient, while serving in the Royal Air Force, was employed with a number of others in unloading gas projectiles; some of the projectiles had been damaged in transit and several of the men received splashes of liquid mustard gas on their clothes. They immediately removed all their clothes with the exception of the patient illustrated here, who kept his trousers on.

Fig. 210 also depicts a typical reaction to mustard gas vapour. The circumstances of the exposure in this case are not altogether without humour: a

FIG. 209.—Shows the typical reaction to liquid mustard on damp areas of the body, such as the groin, perineum, etc. (*Photograph by Percy Hennel.*)

number of soldiers who were housed in Nissen huts in a cold north-east area of Scotland during the winter obtained by some unexplained means a container of what they imagined to be varnish, but was in fact liquid mustard gas. Despite their miserable surroundings they expressed the aesthetic side of their natures by varnishing the stove in their hut. That evening they sallied forth and had a 'celebration', only returning to the hut at a late hour of the night. In their own words "the paint smelt a bit", but because of the extreme cold they closed all the windows. They awakened with a mustard reaction, but without the usual blistering.

According to the classification of mustard-gas burns of the eyes (Ida Mann) into four degrees of severity, the case illustrated here, by reason of the absence of corneal haze, of profuse lacrimation, and of photophobia, belonged to the mildest category.

Along yonder wall of Room III within our College these mute specimens with which this Lecture deals, arranged in their silent rows and designated by mere numbers to-day, yet constitute a visible symbol of British courage and hardihood, making an appeal to men and women of British bone and blood to keep faith with those who perished within our fortress island, all over the Seven Seas, in lands across the waters that wash our own shores, in the jungle and the desert, the atolls and the ice-fields, and in the high-up challenging skies, to decry and defy the deification of insane and brutish force, the machinations of a counterfeit communist ideology, the lusts of the Soviet hierarchy for territorial aggrandizement and world supremacy, and to maintain those freedoms laid down by the late Franklin D. Roosevelt in the Atlantic Charter.

It may be prophetic, it certainly is apposite, that this Lecture which commemorates a great scientific physician, the patron figure of a sister Royal College, should be delivered in this place which John Hunter's spirit still pervades, surrounded by ruins of Hun violence, yet rising like Phœnix from the ashes. The following well-known lines from one of Shakespeare's sonnets are not inappropriate as a tribute to the great Harvey and Hunter alike :—

> "Though wasteful war shall statues overturn,
> And broils root out the work of masonry,
> Nor Mars his sword nor war's fierce fire shall burn
> The living record of your memory.
> 'Gainst Death itself and all-oblivious enmity
> Shall you pace forth. Your fame will still find room
> Even in the eyes of all posterity
> That wear this world out to the ending doom.
> So till the judgement that yourself arise
> You live in this and dwell in lovers' eyes."

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FIG. 210.—Shows the typical reaction to mustard gas vapour; there is no blistering, but some conjunctivitis without corneal 'haze'. (*Photograph by Percy Hennell.*)

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THE DIAGNOSIS AND TREATMENT OF VASCULAR DISEASES*

WITH SPECIAL CONSIDERATION OF CLINICAL PLETHYSMOGRAPHY AND THE SURGICAL PHYSIOLOGY OF THE AUTONOMIC NERVOUS SYSTEM

BY R. H. GOETZ

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ON THE TREATMENT OF PERIPHERAL VASCULAR DISEASES

1. Physiological Considerations.—The most important function of the circulation is to provide for the nutrition of the tissues. The metabolic needs of the skin amount to 1–2 c.c. of blood for 100 c.c. of tissue per minute. This indeed is a very minute amount. In contrast we have shown that the blood-flow through the same skin in the normal subject during full dilatation may reach as much as 90 c.c. per minute. Similarly, the rate of blood-flow to the extremity as a whole during full dilatation has been calculated as 25 c.c. per minute for 100 c.c. of tissue.

From these figures we can draw two important conclusions: First, the blood-flow through the skin serves other than the needs of the nutrition of the tissue; Secondly, the arteries supplying the skin have to be practically occluded in order to harass the nutrition of the skin. We realize that any patient who develops vascular disease but has no initial symptoms such as intermittent claudication and starts off with skin lesions only, is one whose prognosis we have to view with the gravest concern.

Any interference with the blood-supply will first manifest itself when there is an additional demand upon the blood-supply as created by increased function. In muscles this gives rise to intermittent claudication and already a moderate degree of interference with the arterial blood-flow affecting the vessels to the muscles will produce symptoms. Yet patients fail only too frequently to take any note of the early symptoms and consult their practitioner only when the condition is markedly advanced. In the skin there is no equivalent to intermittent claudication which would direct the patient's attention to the early lesion. Increase in the surrounding temperature, however, increases the cutaneous metabolism and creates an increased demand upon the blood-flow. In the normal subject this demand is satisfied by the dilatation of the vessels. In patients with marked organic arterial disease the occlusion prevents dilatation, and the application of heat results in tissue anoxæmia and consequently is harmful. Indeed, such patients will remark that their pain is increased by local application of heat, either in the form of a hot box, in the hot bath, or by hot-water bottles, and gangrene often occurs following the time-honoured use of such appliances —not despite, but because of, their use (*Fig.* 211).

What degree of diminution in blood-flow can we expect from a given degree of interference with the arterial lumen? Such knowledge is of great importance in forming a clear picture of the pathological significance of the clinical findings and in giving a prognosis. According to Poiseuille's law, which has been elaborated for rigid tubes but has been shown to apply, when slightly modified, to the human circulation as well, the rate of blood-flow varies with the fourth power of the diameter of the vessel. Thus an organic occlusion which decreases the diameter of the lumen by half decreases the blood-flow to about one-sixteenth of the original figure. Only thus is it conceivable that the blood-flow through the skin may be regulated within such wide limits, i.e., between 1 and 90 c.c. per minute. Poiseuille's law, too, enables us to visualize that very little advance in the pathology is required in a case

* Being the second part of the Arris and Gale lectures delivered before the Royal College of Surgeons, London, Jan. 14, 1949.

of organic arterial disease to produce a sudden and dramatic deterioration in the blood-flow seen in so many patients for no apparent reason and often with such catastrophic results.

As suggested, the blood-flow through the skin of the extremities is mobilized for functions other than tissue metabolism. The digital blood-flow serves the dissipation and conservation of heat; the

FIG. 211.—Feet of a boy 3 years of age who suffered from Raynaud's phenomenon in all extremities. During an attack his feet were exposed to heat in order to warm them and gangrene resulted.

extremities are, so to speak, our radiators. In the individual who is under the necessity of preserving heat, the skin vessels are constricted, allowing, as a rule, only sufficient blood-flow for tissue metabolism. The subject who has to dissipate heat presents with dilated cutaneous vessels, facilitating the loss of heat from the skin.

Patients suffering from Raynaud's disease relate that their extremities are continuously cold, even in the absence of definite attacks. Indeed, the blood-flow between the attacks, measured plethysmographically, is not normal, but markedly diminished, and just enough for tissue metabolism (*Fig.* 212). As can be proved, this decrease is due to a high central vasomotor tone, the result of the necessity of the body to preserve heat. Similarly, in hypothyroidism and anorexia nervosa, where the metabolism is greatly diminished and heat production minimal, we find the same increase in central vasomotor tone and occasionally Raynaud's phenomenon may be observed. The opposite—namely, an increase in the cutaneous blood-flow—is observed in such conditions where the metabolism or heat production is above par, for instance, hyperthyroidism, fevers, and during pregnancy.

Although such clear-cut relations between cutaneous blood-flow and metabolism are observed, it is notoriously difficult to influence therapeutically vasospastic attacks or the pathologically high vasomotor

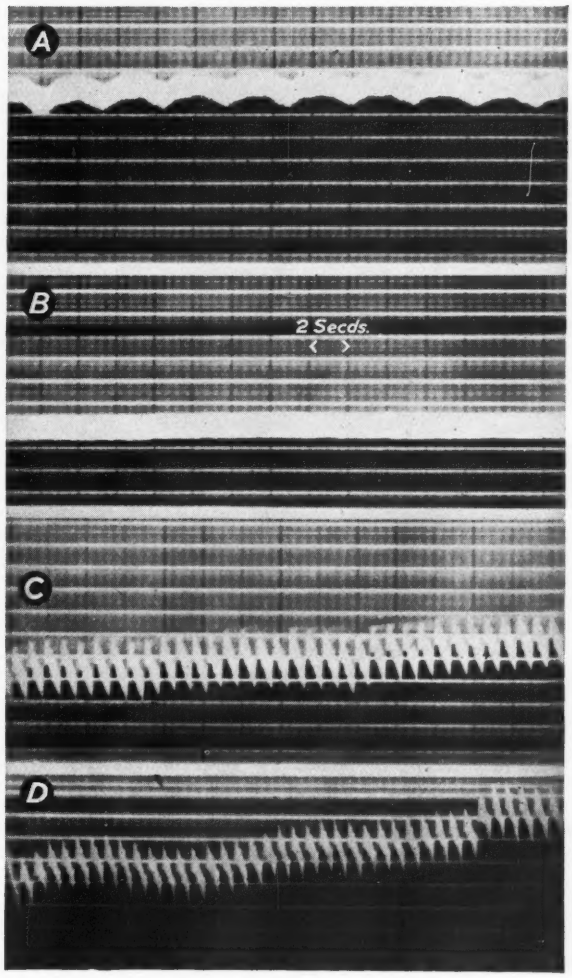

FIG. 212.—Plethysmogram (second left finger) of a woman 23 years of age who suffered from Raynaud's phenomenon. A, Between attacks the hands were cold but normal in colour. Pulse volume hardly registrable. B, During an attack. Pulse volume completely absent. C, Fully dilated after 30 minutes' body heating. Pulse volume ± 0·012 c.c. D, After additional immersion of hand in water of 42° C. for ten minutes (local heat). No further increase in the height of the pulse volume. White horizontal lines (calibration for pulse volume) = 0·01 c.c.

tone by measures aiming at increasing metabolism and thus forcing vasodilatation. I am referring to the administration of thyroid extract and similar drugs in cases of Raynaud's phenomenon and acrocyanosis.

The arterial tree is not a rigid anatomical structure, but a functional entity readily adaptable to changes in its surroundings, both intrinsic and extrinsic. What holds good for the skin vessels may not be true for the vessels supplying the muscles and indeed one and the same stimulus is often followed by opposite changes in skin and muscle vessels. In addition, changes in the vasomotor tone of the vessels in one area may indirectly affect the blood-flow through another. Thus constriction of the cutaneous vessels decreasing the blood-flow

through the skin may indirectly cause an increase in blood-flow through the muscles. Another example of this is seen in the behaviour of the blood-flow through the sympathectomized limb on body heating (*Fig.* 213). Both the pulse volume

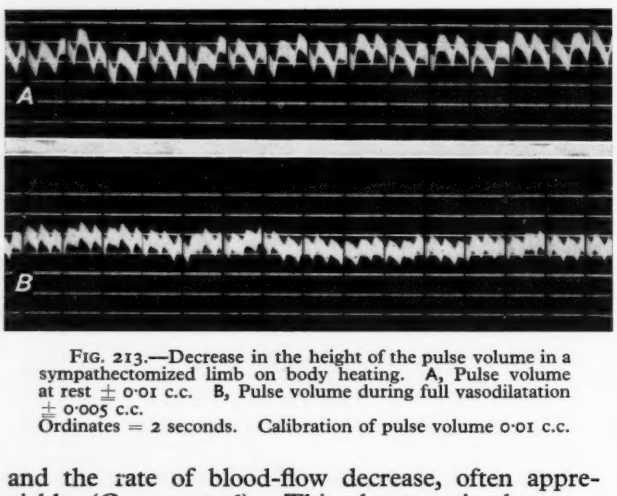

FIG. 213.—Decrease in the height of the pulse volume in a sympathectomized limb on body heating. A, Pulse volume at rest ± 0·01 c.c. B, Pulse volume during full vasodilatation ± 0·005 c.c.
Ordinates = 2 seconds. Calibration of pulse volume 0·01 c.c.

and the rate of blood-flow decrease, often appreciably (Goetz, 1946). This decrease is due to a shunting of blood away from the sympathectomized extremity: as body heating releases the vasomotor tone in the non-sympathectomized vessels, they offer less resistance to the blood-flow. At the same time the tonus in the sympathectomized vessels remains unaltered and actually now appears higher when contrasted with the remaining vessels. The blood, in consequence, follows the path of least resistance, and is shunted away from the sympathectomized limbs during body heating. The practical implication of this is obvious. Diathermy and other reflex dilatation methods diminish the blood-flow in the sympathectomized limb and consequently are contra-indicated!

Another example is provided by the sudden appearance (first reported by us in 1946) of Raynaud's phenomenon in the upper extremities in patients who have undergone a Smithwick operation for hypertension (Goetz, 1947). It seems that the phenomenon is seen particularly in those cases in which there is a definite reduction in blood-pressure following the procedure. Raynaud's phenomenon occurs because the vessels of the hands are under a high neurogenic vasomotor tone, which before operation was counteracted by the increased blood-pressure. Once the systemic blood-pressure is lowered this high vasomotor tone will be able to produce a greater degree of vasoconstriction, which in some cases leads to Raynaud's phenomenon. The phenomenon does therefore not occur in the lower (sympathectomized) extremities. If Raynaud's phenomenon occurs post-operatively it suggests that in that patient the vasomotor system is implicated and hypertension is most likely neurogenic as against humoral in origin.

This occurrence of Raynaud's phenomenon is one reason why we recommended extension of the operation to include D.2 in order to obtain sympathectomy of the upper extremity.

The blood-vessels through the skin and the muscles are often affected in varying degrees by the same pathological process. Some patients develop threatening gangrene without ever having experienced intermittent claudication. We have already pointed out the reason why the prognosis in these cases as a rule is grave. The opposite, i.e., intermittent claudication with a relatively good cutaneous circulation, is more common, and offers as a rule a much better prognosis for the limb. The latter case will seek the advice of his doctor at an earlier date as far as the cutaneous circulation is concerned. It is from a deficiency of the blood-flow through the skin that the danger to a limb arises.

The metabolites developing in normal and ischæmic muscle during exercise produce by direct action a dilatation of the blood-vessels supplying the muscle. Therefore the degree of exercise tolerance, i.e., the degree of intermittent claudication experienced by the patient at successive tests, is very constant (Goetz, 1942). Normally these metabolites are washed away by the increased arterial blood-flow. With organic arterial disease dilatation can be but minimal and the metabolites accumulate, producing increasing pain and forcing the patient to rest. Barcroft and his colleagues have demonstrated that the increase in muscle blood-flow resulting from sympathetic paralysis is minimal and only about one-thirteenth of that produced by the action of muscle metabolites. This explains why sympathectomy cannot be expected to produce a dramatic improvement in the exercise tolerance or intermittent claudication. In our experience an actual, but temporary, decrease in the exercise tolerance has been observed in many a case, which may be due to directing the blood away from the muscles to the dilated blood-vessels of the skin. This, however, is no reason for advising against sympathectomy in these cases. In the suitable case there results an improvement in the circulation of the limb in general in which the blood-flow through the muscles takes part eventually and the exercise tolerance improves.

2. On the Adrenaline Sensitivity of Sympathectomized Vessels.—Sympathetic denervation of the lower extremities has never presented any problem, in contrast to sympathectomy for the upper limbs, which, despite numerous modifications of the original procedure, has not given the ideal results so easily achieved in the lower limbs.

However, I should like to point out, before proceeding further, that although the indications for sympathectomy naturally are the same for the upper and lower extremities, sympathectomy of the lower limbs is more commonly performed to release superadded spasm in cases of organic arterial disease, whereas in the upper limbs cases of Raynaud's phenomenon without organic involvement constitute the majority. Evidence at our disposal has shown that this difference may be of some importance on account of a possible local fault involved in Raynaud's phenomenon. In comparing the results of the operation in the upper extremity with those in the lower, we should therefore select only cases in which the operation was carried out for the same reason, i.e., Raynaud's phenomenon. On the other hand, much will be learned from comparing the results of cervicodorsal sympathectomy in such conditions as acrocyanosis, Raynaud's disease,

and other vasospastic disorders with cases in which the operation was performed other than for vascular disease. Indeed, it is our impression that cervicodorsal sympathectomy fails more commonly in cases of Raynaud's disease, and that if the procedure is carried out for non-vascular conditions it is more satisfactory. The report by Haxton (1948) on cases of hyperhydrosis would support this view. In the past many authors have lost sight of the fact that by carrying out a sympathectomy all we can hope for in a case of Raynaud's phenomenon is to abolish attacks which are induced by central vasomotor impulses. After all, that is the most sympathectomy will abolish. Factors which before operation induced attacks by extrinsic mechanisms, such as the local application of cold or the outpour of adrenaline, will still be operative after sympathectomy. Therefore, the fact that a certain patient still complains of attacks of Raynaud's phenomenon after sympathectomy does not mean that the operation failed to sympathectomize the vessels or that regeneration took place. Nor does it mean that there has been reorganization of sympathetic pathways. Such inference has to be carefully investigated. Attacks are observed in fully sympathectomized vessels, sometimes within a week of operation.

Several theories have been advanced in the past to explain the poor results following sympathectomy in cases of Raynaud's phenomenon. After Kuntz's nerve had been described they were attributed to undivided fibres arising in the second thoracic sympathetic ganglion innervating the brachial plexus, and the original operation (stellatectomy) was extended to include the second ganglion. This *apparently* did not materially alter the results. It was then that, based on the experimental work of numerous authors, sensitization to adrenaline was suggested as the cause of the clinical relapses. As animal experiments showed that postganglionic operation rendered the structures three times more sensitive to adrenaline than the preganglionic one, the latter type of operation was advocated. It was then that Telford (1935) and Smithwick (1936) demonstrated that the arm can be completely denervated by dividing the preganglionic rami of the second and third thoracic ganglia and cutting of the sympathetic trunk below the third ganglion (preganglionic ramisection). The immediate results of this operation appeared superior to those following cervicodorsal sympathectomy. The late results are disappointing, however, and still do not compare favourably with those following denervation of the lower extremities.

Although at the time various authors, notably Fatherree and co-workers (1938), demonstrated that there was little difference in the adrenaline sensitivity whether the operation was pre- or postganglionic in type, the preganglionic ramisection came to stay in favour of the cervicodorsal ganglionectomy, in my opinion mainly because it does not produce a Horner's syndrome, and not for its supposed merits in producing permanent sympathetic paralysis.

Two possibilities were then explored: First, regeneration of interrupted fibres; and, secondly, functional reorganization of undivided fibres within the intact cervicodorsal chain and ganglia.

Smithwick, believing in the former, recommended intraspinal root section of T.2 and T.3 and cutting of the trunk below the third paravertebral ganglion, as well as covering of the mobilized trunk with a cylinder of silk or cellophane in order to prevent regeneration. Finally, the free lower end of the trunk was transplanted into the muscle of the wound. White and Hamlin (1945) recently suggested the use of tantalum instead of the silk cylinder. Even such procedures did not prevent the recurrence of vasoconstrictor tone, and a "fantastic" and an "uncanny power of regeneration" (Haxton, 1947) appeared to be the only reasonable explanation to many authors.

Everybody accepts the contention that it is the preganglionic fibres which regenerate and reinervate the ganglion cells in the stellate ganglion. However, it appears difficult to imagine—and this point has so far not been acknowledged in the literature—that regeneration should take place following removal of the ganglion cells, i.e., after cervicodorsal ganglionectomy. Not only are there no ganglion cells for the preganglionic fibres to innervate, but in addition the preganglionic fibres are cholinergic, while the vasomotor fibres are adrenergic. Consequently the two will not link up with each other. Preganglionic ramisection is therefore the operation more liable to regeneration as compared with ganglionectomy, as the ganglion cells and their postganglionic fibres are left intact for the preganglionic fibres to re-establish synapsis. Indeed, in our experience it is only the preganglionic ramisections in the upper extremities in which the vessels have regained reflex vasomotor control, while in the cervicodorsal ganglionectomies they remained permanently sympathectomized. In one of our cases* the ganglionectomy was performed as long ago as 1917 and the limb is still fully sympathectomized to-day. Others had their cervicodorsal ganglionectomies ten to fifteen years ago and do not show any sign of regeneration.† In contrast to Haxton, we do not find that the myosis of Horner's syndrome has disappeared in these cases, nor are sudomotor fibres demonstrable. Provided, therefore, that adrenaline sensitivity does not play the role ascribed to it in the past, cervicodorsal ganglionectomy, apart from the fact that it is associated with a Horner's syndrome, should be the operation of choice. We therefore have re-examined the question of the adrenaline sensitivity, employing more sensitive methods of recording the blood-flow.

Using our plethysmographic method (Goetz, 1949) and a photo-electric drop recorder (*Fig.* 214), the blood-flow in twenty-seven limbs was examined at various intervals following the various types of operation. Our method permitted us to compare the result of the sympathectomized limb with the opposite normal or in some cases in which two different types of operation had been performed the results in both could be conveniently compared. *Fig.* 215 represents a typical example of a test in a

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* The surgeon who performed the operation is, unfortunately, not known to us. It was performed at the South African Army Hospital stationed at the time at St. Thomas's Hospital, London. See also *Figs.* 63–65 in the first part of this paper (p. 34).

† These cases were operated on by Professor C. F. M Saint, who kindly permitted their study.

patient who had undergone cervicodorsal ganglionectomy on the right side and a preganglionic ramisection on the left for Raynaud's phenomenon. Adrenaline 1–500,000 was given by the intravenous drip method previously described (Goetz, 1948, a) and each drop was recorded in the tracing as it fell through the drip. As can be seen, before adrenaline was given the pulse volume in the right limb was 0·02 c.c. and that of the left one only 0·012 c.c. From previous examination, we knew that there was no organic arterial disease present. The vessels in the left limb had regained, therefore, a higher but not central tone. As can be seen, it took thirty seconds before any reaction of the vessels was recorded, by which time thirty drops of adrenaline had been administered.

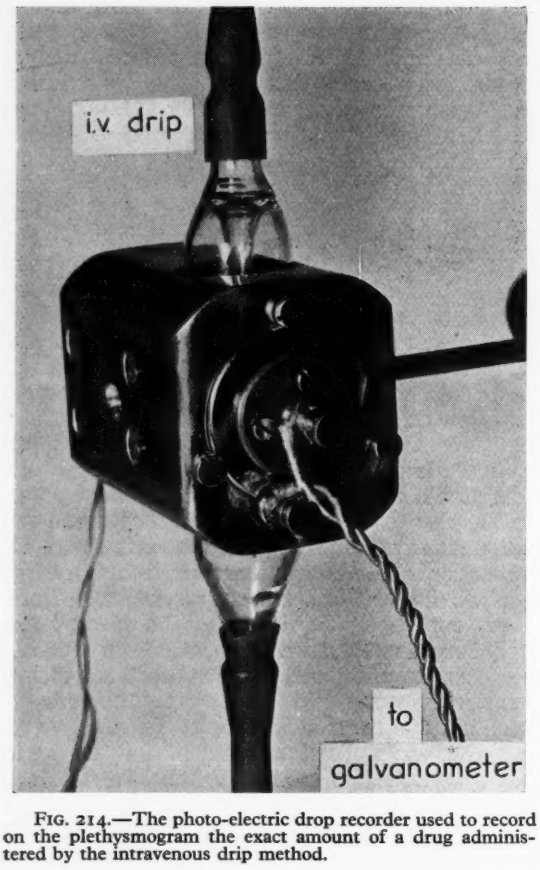


FIG. 214.—The photo-electric drop recorder used to record on the plethysmogram the exact amount of a drug administered by the intravenous drip method.

There was no material difference in the reaction of the vessels in the two limbs to the drug, although at the height of the reaction the pulse volume in the right was slightly larger than in the left limb, the proportion being, however, similar to the findings before administration of the drug. In neither limb did the vessels go into spasm arresting the blood-flow as is seen in Raynaud's phenomenon.

Fig. 216 was obtained in a case in which a cervicodorsal ganglionectomy had been performed on the left side, the other limb being normal. When the injection was started the pulse volume in the normal limb, which was fully dilated, reached ±0·032 c.c., that in the sympathectomized limb ±0·018 c.c. As in the first case, it took thirty seconds before any effect was registered. Once adrenaline started to contract the vessels, the fall in the digital volume appeared to be actually greater in the normal limb than in the sympathectomized one. However, we have to take into account the fact that the vessels were more dilated at the outset and therefore constriction would produce a more powerful effect. As regards the height of the pulse volume, this decreased in the normal from ± 0·032 to ± 0·012 c.c., while that of the sympathectomized limb decreased from ± 0·018 c.c. to ± 0·0007 c.c. The reaction did not last any longer in the sympathectomized limb than in the normal and *Fig.* 216 demonstrates that two minutes after the last drop both the pulse volume and the digital volume are again at their original height.

Further tests performed on the same patient with the vessels of the sympathectomized limb completely relaxed by the local application of heat, so that the pulse volume was equal on both sides, did not reveal any substantial difference in the reaction of the two limbs. Even doses which produced marked untoward effects on the heart and respiration did not alter the vascular reaction in the sense of a hypersensitivity of the vessels to adrenaline.

It may be argued that the adrenaline circulating in the body and causing the hypersensitivity of the

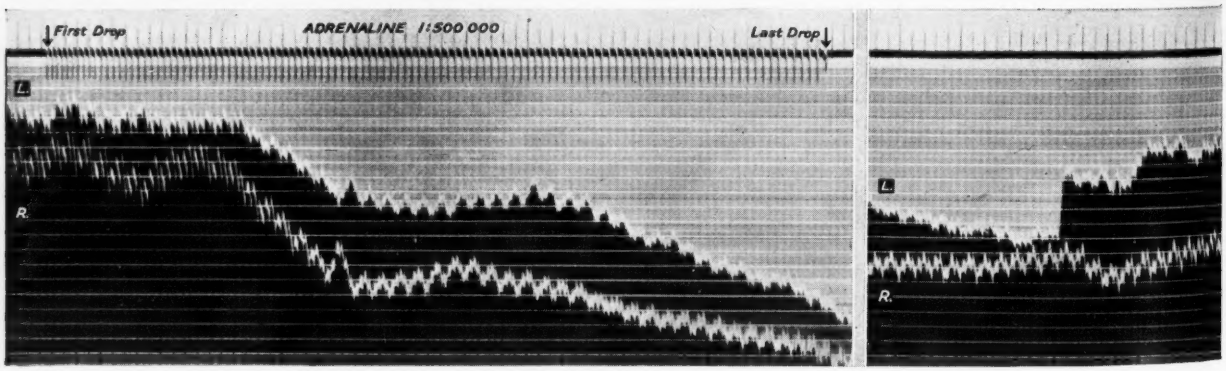


FIG. 215.—The effect of intravenous adrenaline on the blood-flow in the two upper extremities simultaneously recorded. Patient had undergone a dorsal sympathectomy (removal of T.2 to T.4—preganglionic sympathectomy) on the left side, and a cervicodorsal sympathectomy (removal of the middle cervical ganglion, the stellate and second and third dorsal ganglia—post-ganglionic sympathectomy) on the right. Note the drop recording on the top of tracing.

Ordinates = 2 seconds. White horizontal lines: calibration for the plethysmogram. A movement from one line to the next represents a change in blood-flow of 0·01 c.c. At the interruption of the tracing the menisci were adjusted.

vessels is different from the adrenaline just administered. Therefore we tested nineteen of our patients by giving 20–25 units of insulin intravenously so as to produce marked hypoglycæmia and all the symptoms and signs of an outpour of adrenaline. *Fig.* 217 illustrates such a test in the same patient in

Our investigations, therefore, do not support the concept that hypersensitivity to adrenaline is responsible for the clinical relapse in cases sympathectomized for Raynaud's phenomenon. If sensitization of the blood-vessels does occur, it is not of any clinical significance. Fatherree and co-workers

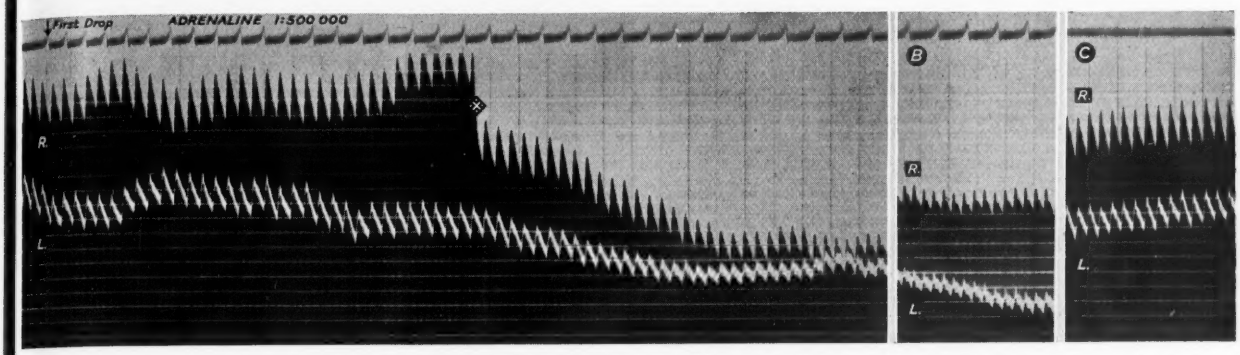


FIG. 216.—The effect of intravenous adrenaline on the peripheral blood-flow in the two upper extremities of a patient who had undergone a cervicodorsal sympathectomy (removal of stellate plus second dorsal ganglion) on the left side thirty-one years ago. Note drop recording on the upper border of the tracing. At * adjustment of the plethysmogram of the right limb. B, Taken at the height of the reaction and at end of infusion. C, Two minutes after the last drop of adrenaline had been given. Note complete recovery. (For explanation *see* text.)
Ordinates = 2 seconds. White horizontal lines = 0.01 c.c.

whom *Fig.* 216 was obtained. Before the test the pulse volume in the normal limb was ±0·023 c.c., that of the sympathectomized side ±0·013 c.c. At the height of the reaction the pulse volume in the sympathectomized limb decreased to ±0·005 c.c., while that in the normal had come down to ±0·01 c.c. There seems indeed a change in favour of the normal limb in this case which, in other cases could not

have already expressed the opinion that in man the removal of the ganglia does not produce a sensitization nearly as great as in the monkey, in which, in turn, it appears not to be as great as in the cat. No doubt the indiscriminate application to man of results obtained in animals has not only confused, but far too often misled, us in the past in our understanding of the autonomic nervous system in man.

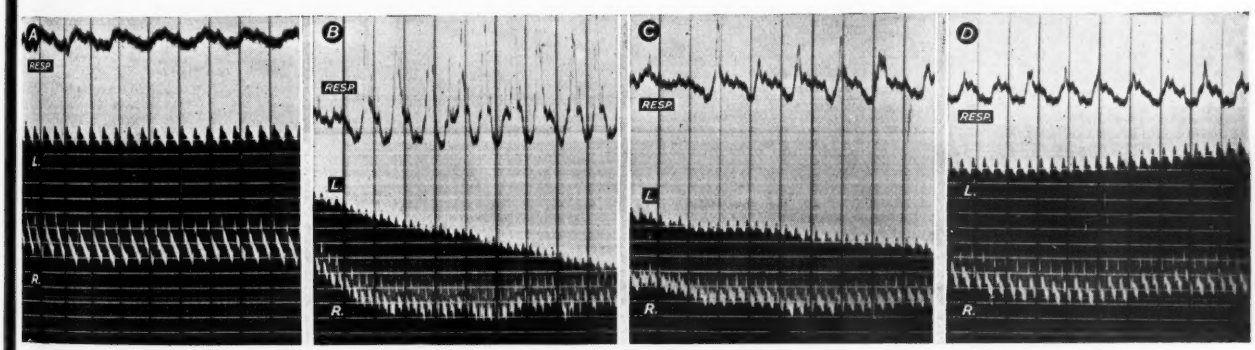


FIG. 217.—Effect of 25 units insulin given intravenously on the peripheral blood-flow of both upper extremities simultaneously recorded in a patient who had undergone cervicodorsal sympathectomy on the left side thirty-one years previously (same patient as *Fig.* 216). A, Before injection of insulin. B, At the height of the vascular reaction. Note tachycardia and tachypnœa. C, Immediately after glucose had been given. Note respiration has already returned to normal. D, Twelve minutes after B and six minutes after C. Return to normal.
Ordinates = 2 seconds. Calibration = 0·01 c.c.

be confirmed. However, at no moment was there any complete closure of the peripheral vessels as we see it with Raynaud's phenomenon. The dose in this case was rather a large one, producing very severe symptoms, as can be seen from the marked tachypnœa and hyperpnœa.

Fig. 218 refers to a case in which a lumbar sympathectomy had been performed on the left side (*Fig.* 218 D–F). Twenty-five units of insulin were given intravenously and the cuttings from the original film show that at the height of the hypoglycæmia the pulse volume was no more diminished in size on the operated side than on the normal.

Geohegan and Aidar suggested that recovery of reflex sympathetic activity after preganglionic ramisection of the upper limbs is due to reorganization of sympathetic pathways, made possible by the fact that we leave the outflow from the first dorsal segment intact. Normally this does not carry sympathetic fibres to the limb but to the face and neck only. Following ramisection of D.2 and D.3 and the chain below D.3, reorganization takes place and the preganglionic fibres reaching D.1 now take over the innervation of the ganglia deprived of their preganglionic pathways but still intact. They proved this point, however, only in the cat. The possibility

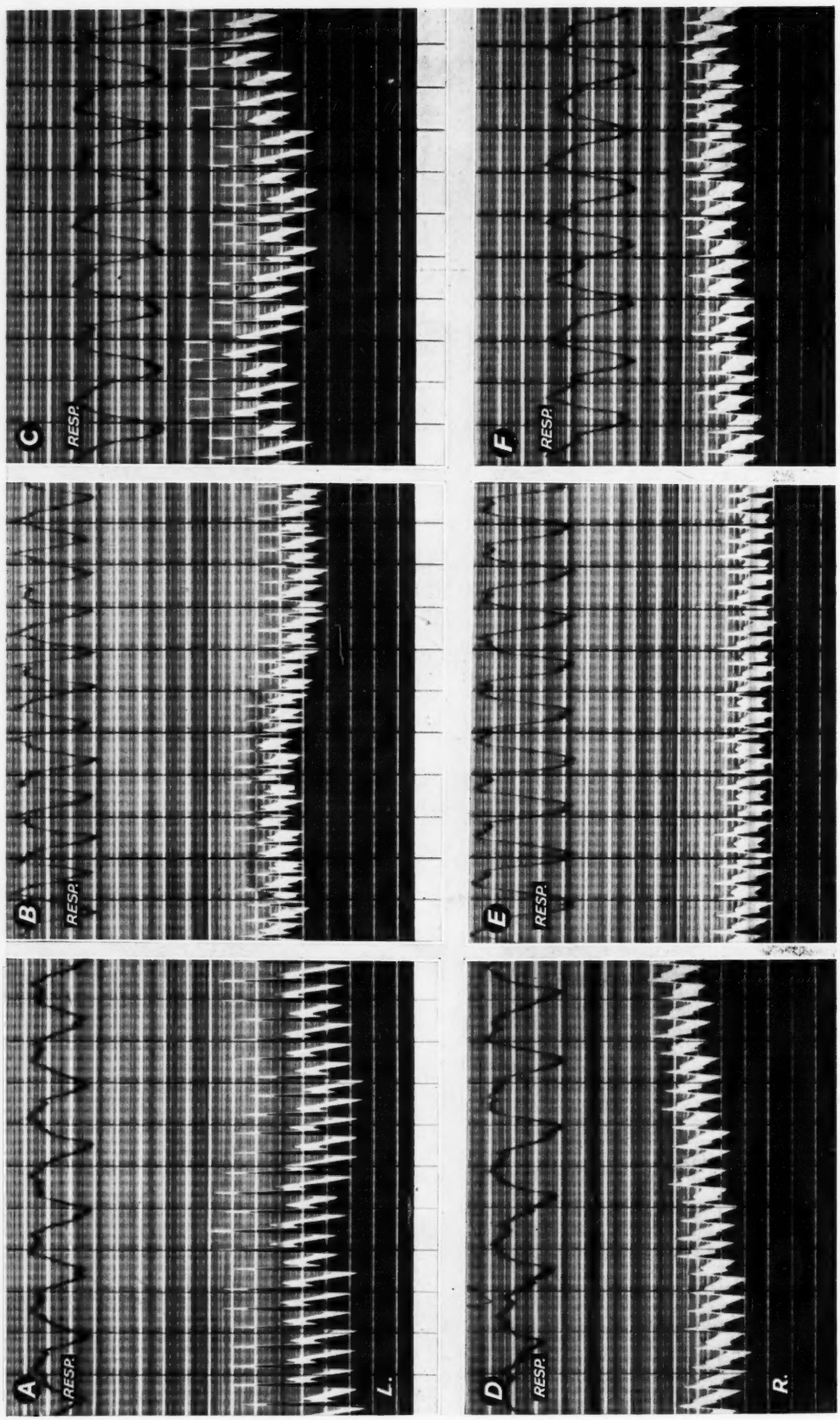


FIG. 218.—Effect of 25 units insulin given intravenously on the peripheral blood-flow in a patient who had undergone a lumbar sympathectomy on the right side (removal of L.2 and L.3) fourteen years previously. A and D, Before the injection of insulin. B and E, At the height of the hypoglycæmia. Note the marked increase in respiration and heart-rate. C and F, Forty-two minutes after the injection of insulin. Complete recovery.
Ordinates = 2 seconds. Calibration = 0·02 c.c.

of reorganization is an added complicating factor inherent in the preganglionic ramisection only.

Having demonstrated that sensitization of the blood-vessels to adrenaline is not of any clinical significance, the necessity of insisting on a preganglionic ramisection falls away. There is, therefore, no reason warranting the greater risk of regeneraion of the preganglionic ramisection and the possibility of reorganization. The most important point, i.e., lasting and complete sympathectomy, can best be achieved by performing ganglionectomy, and at Groote Schuur Hospital we have therefore reverted to the cervicodorsal ganglionectomy and if possible include the middle cervical and third dorsal ganglia as well in order to be certain that there remain no undivided preganglionic fibres and postganglionic synapsis as in the old stellatectomy. Thus will we be certain that complete sympathectomy of the upper limb will be achieved. The much-feared Horner's syndrome has not worried our patients, particularly when the operation was performed bilaterally. It appears, too, that the inclusion of the middle cervical ganglion does somehow minimize the droop of the lid, which is the only part of Horner's syndrome worrying the patient from the cosmetic point of view.

3. The Treatment of Peripheral Vascular Diseases.—This may be divided into three main groups—namely, prophylactic, conservative, and operative. In the course of this lecture it is naturally impossible to deal with every type of treatment devised in the past and in vogue at present. However, I would like to touch on a few of the more important points where we differ from what you will find in the standard text-books and about which colleagues have repeatedly consulted me during my short stay in London.

Physicians are, as a rule, reluctant to advise surgery, as indeed are surgeons. Consequently, conservative treatment is in many cases persisted in for too long a period. It is my belief that through prolonged conservative treatment and for sentimental reasons, surgery is far too often postponed and thus the chance of saving the limb often thrown away. I am referring particularly to such operative procedures as sympathectomy, débridement, and nerve section.

a. Sympathectomy.—If a patient comes to us with symptoms referable to peripheral vascular disease and in whom we diagnose such a condition, it is our first duty to ascertain by special tests whether he is a suitable subject for sympathectomy. For this plethysmography is particularly suitable, as I have already pointed out. The degree of spasm can be determined, the part played by the organic obstruction assessed, and the amount of collateral circulation measured. It is especially the last-named which is of such paramount importance. A case may present as an organic arterial disease, skin temperature readings may suggest complete organic occlusion, and yet plethysmography may be able to prove, when skin temperature readings fail, that by ablation of central sympathetic tone the collateral circulation can be released and improved to such an extent as to make it well worth while proceeding with sympathectomy.

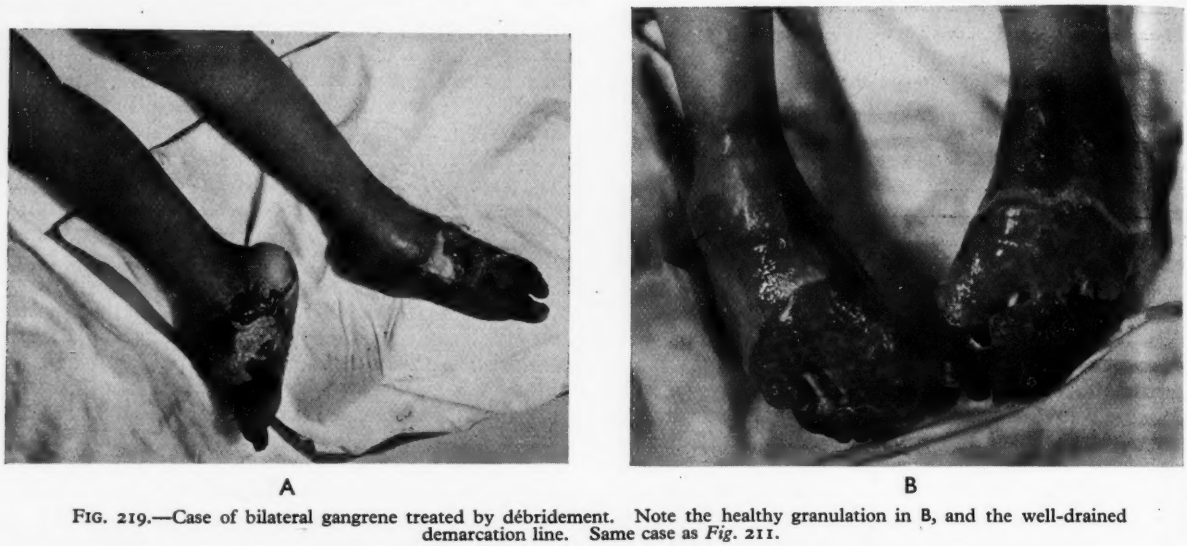
Indeed, some of the controversial results reported from various clinics in cases of organic occlusion following the treatment by sympathectomy are attributable to the different degree of the development of the collaterals in cases of organic arterial disease, which could not be assessed by the methods employed by the respective authors. By carrying out plethysmography as a routine measure we can pick out those cases of organic occlusion which will respond to sympathectomy. As there is the tendency to sympathectomize any and every patient, I have to point out that in those cases of complete occlusion with poor collateral circulation in which sympathectomy appears to be contra-indicated as judged from plethysmography, we have found that operation not only failed to bring about improvement, but merely prolonged the patient's tortures, postponed amputation, and increased hospitalization.

Sympathectomy should be carried out early and not as a last resort. Sympathectomy is no longer a serious operation carrying a high mortality. In Cape Town we have carried out well over 400 sympathectomies of all types (excluding splanchnicectomies) without a single operative death and the operation is performed by the general surgical staff using the retroperitoneal approach in the case of the lumbar sympathetic and the anterior or the transthoracic (through the second intercostal space in the axilla) approach in the case of the cervicodorsal sympathectomy. It has to be realized that by increasing the peripheral blood-flow and by releasing any superadded spasm the pathological process cannot be reversed, but there can be no doubt that in many cases the disease takes a completely different course and gives the collateral circulation a chance to develop. Our results have been extremely good and we have cases of organic arterial disease in our records in which the blood-flow years after sympathectomy has eventually returned to normal.

I am fully aware that when discussing this subject I find myself in sharp contrast to the teaching of some of the most prominent American colleagues in this field. To quote their experience : " These operations (ganglionectomy and sympathectomy) have no place whatsoever in the treatment of gangrene or in any phase of thrombo-angiitis obliterans ". Or passages like this : " The magnitude of the procedure and the great operative risk, as well as a fairly high mortality, certainly do not justify its performance in either thrombo-angiitis obliterans or arteriosclerosis ". Silbert, of New York, who is said to have the vastest experience in vascular diseases and whose judgement must command the highest respect, does not subscribe to the value of sympathectomy. As I have just pointed out to you, this is in sharp contrast to our experience. However, I have to stress once more that all cases must be carefully selected and scientifically assessed. The operation must be clearly indicated. Its indiscriminate use in every case of arterial disease is to be deplored and can only result in the verdict of these prominent American scientists. No one would open the chest and operate for a ductus or coarctation without having performed the necessary pre-operative investigations. No one should perform a sympathectomy without having first assessed the peripheral circulation by adequate tests. We have seen gangrene occurring in limbs while the patient is recovering from a sympathectomy that was clearly not indicated, the gangrene being

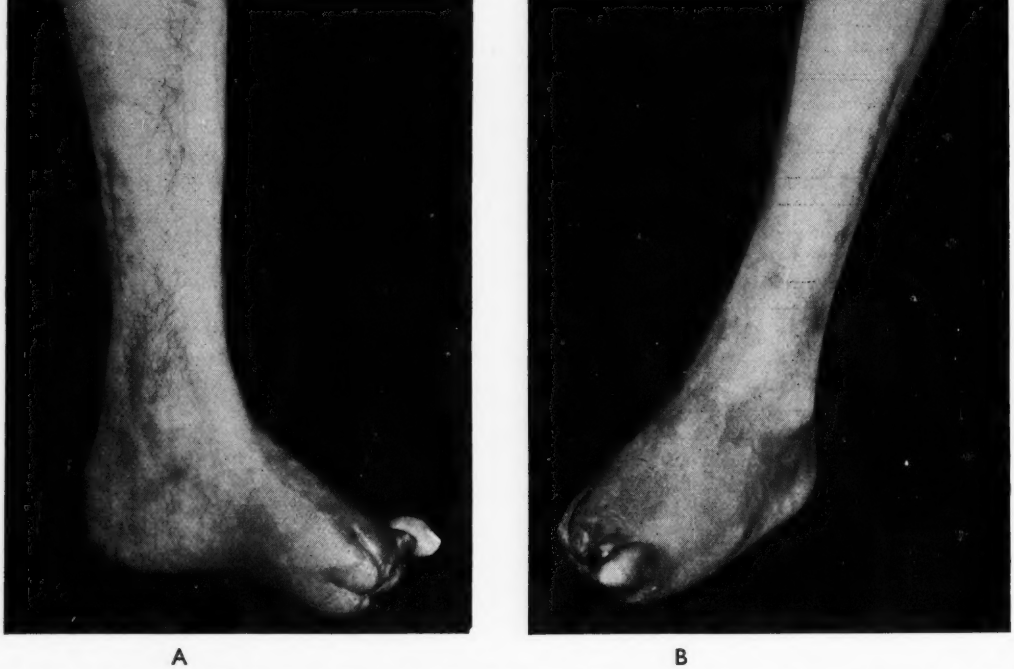
precipitated by the general depression that follows any operation.

b. Débridement.—The next point I mentioned was débridement. A limb with a gangrenous patch is, as a rule, viewed as 'untouchable'. The surgeon's greatest joy is to see a gangrene which can be called 'dry', and it is the aim to convert any so-called 'wet' gangrene into the dry modification by applying alcohol dressings. This is achieved in most cases with these dressings. By coagulating the superficial gangrenous tissue, the alcohol mummifies the digit and forms a crust which not only irritates but under which the bacteria proceed with their destruction of tissues. Far too often we find large pockets of pus underneath what has been regarded as a perfectly satisfactory dry lesion. In surgery of vascular disease, as in general surgery, adequate drainage is one of the first principles and therefore we decry the use of alcohol as a dressing for gangrene in the above sense. In contrast to this, we recommend daily soaking of the limb for 20 minutes in a soap lather or any antiseptic solution and removal of the dead tissue, if possible, trimming of loose bits of nail without removing the healthy part, opening of blisters and exploration of any possible pockets, followed by the application of an antiseptic or antibiotic ointment.



A B

FIG. 219.—Case of bilateral gangrene treated by débridement. Note the healthy granulation in B, and the well-drained demarcation line. Same case as *Fig.* 211.



A B

Fig. 220.—Leg of a patient suffering from thrombo-angiitis obliterans. The gangrenous area has been treated by débridement and nerve section. The patient had already lost the other leg. He was able to get about very well with this foot three years after the amputation of all but the small toe. Note scars from nerve sections.

It is by following this procedure, I am sure, that we have been able to save many a limb, and could perform local amputation of toes and of the forefoot where we would else have had to sacrifice the lower leg or the entire limb (*Figs.* 219, 220).

c. Nerve Section.—If the patient has marked and continuous ischæmic pain it usually results in utter deterioration of his morale, and after a short time many a patient will grow tired of any treatment and will demand amputation. Before amputating any limb in cases of peripheral vascular disease we should think twice. We must remember that we are dealing with a systemic disease and that in most cases the other limb is affected as well. Even if the patient has no symptoms, the other limb is potentially in danger until we have proved the contrary. Forty per cent of all cases which have been amputated return and require surgery for the other limb within three years. Now in many of these cases the pain is more of a neuritic character—an ischæmic neuritis—rather than due to ischæmia of the tissues *per se*, as can be demonstrated plethysmographically. If, therefore, the blood-flow on plethysmography proves to be sufficient for the nutrition, we do not hesitate to sever the peripheral nerves to the foot in order to alleviate pain and permit adequate treatment (*Fig.* 220). This, again, should not be left as a last resort, but as soon as pain becomes uncontrollable and the necessary tests have been performed, it should be recommended. Rarely have we experienced infection of the operation wounds following this procedure and there remains no disability.

Having attended to the local lesion, controlled the pain, given vasodilator drugs, prohibited smoking, and decided about the question of a possible sympathectomy, intermittent venous occlusion therapy and other mechanical aids are at our disposal, and are being employed increasingly.

d. Intermittent Venous Occlusion Therapy.—No-one seems to have any lucid idea as to the pressures to be employed in a particular case, at what cycles to set the machine, and for how long to proceed with the treatment altogether. Yet its indiscriminate use is strongly to be condemned as it may actually harm the patient.

On general principles, three points have to be considered: First, the pressure should be low enough in order not to interfere with the arterial inflow; secondly, it should be as high as possible in order to give the maximum result; thirdly, if we are dealing with a limb in which the capillaries have been damaged by the ischæmia, venous congestion above a certain point will produce œdema by the escape of fluid through the capillary wall which will interfere with capillary blood-flow. Therefore, the highest pressure which does not interfere with the arterial inflow may not be suitable and may actually be too high.

Most colleagues use the diastolic pressure as a guide. This is not a bad rule if the cuff can be applied in a portion of the limb in which the main vessels are patent and under the systolic pressure. The difficulties arise, however, once we are dealing with occluded main vessels and the circulation is being carried on by collaterals. The pressure in the collaterals may be as low as 40 or 50 mm. Hg, and the diastolic pressure on the arm 80 mm. or more. It stands to reason that a pressure of 60–80 mm. Hg will obstruct the collateral circulation in such cases and therefore be harmful. In order to obtain the maximum beneficial result and to avoid harm, we should be guided by a method able to determine exactly the pressures required.

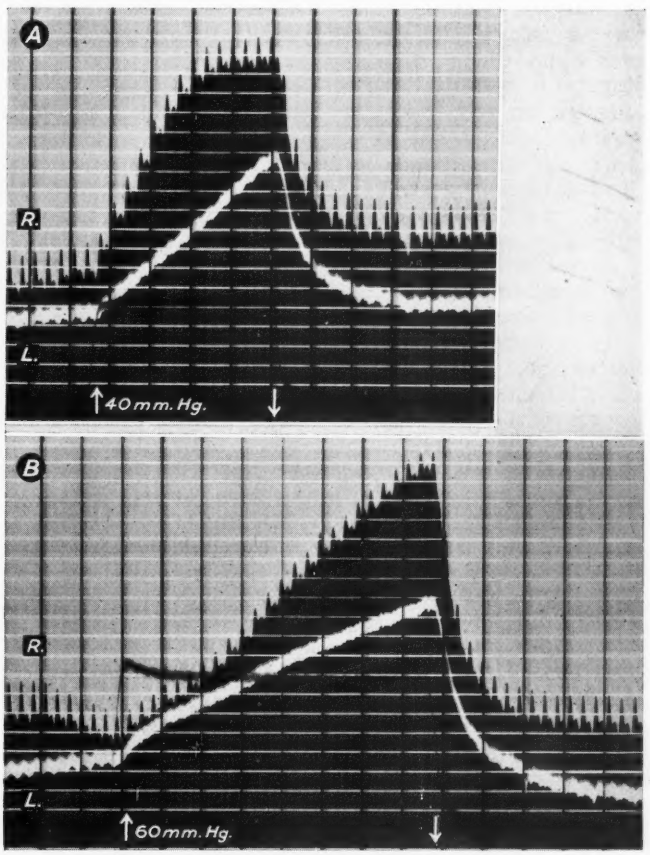


FIG. 221.—Plethysmogram demonstrating the interference with the arterial inflow produced by increasing congestion pressures. In A a pressure of 40 mm. was applied, and in B the pressure was 60 mm. In A, with 40 mm. venous congestion, the plethysmogram of the left leg increases 0·06 c.c. in 6 seconds, but in B only 0·03 c.c. in 6 seconds. The arterial inflow is therefore interfered with by the higher pressure and is actually only half that recorded with the lower pressure. Obviously, 60 mm. would be too high a pressure to be used for intermittent venous occlusion therapy, and would decrease rather than increase blood-flow.

In Cape Town, therefore, all cases which are being put on this treatment are first tested plethysmographically. By means of the venous congestion test we determine exactly the highest pressure which will not interfere with the main or the collateral arterial inflow. Applying successively higher pressures for obstructing the venous return, we note that, as long as the arterial inflow is not interfered with, the same gradient of the tracing is being recorded. As the pressure increases, it interferes with the arterial inflow and the slope of the tracing decreases (*Fig.* 221). By observing the highest pressure not interfering with the arterial inflow, we have an exact method to guide us in applying the correct pressures for this type of therapy.

The venous congestion tests can give us information, too, as to whether the pressure intended to be used is likely to produce œdema. The formation of œdema in the tissue spaces will interfere with capillary blood-flow and therefore will be harmful to the patient. If there is any escape of fluid from the vascular tree into the tissues no equilibrium between arterial inflow and venous return can be established during the application of the venous congestion and the tracing continues to rise. In addition, on release of the venous congestion, the tracing does not fall to the base-line, but remains raised by the amount of fluid which has escaped into the tissue spaces.

By using plethysmography, therefore, we can determine exactly what pressures should be used in each particular case and thus hope to produce the maximum results and prevent any harmful effects to our patients.

e. Hypertonic Saline Injections.—Lastly, I should like to make a few remarks concerning hypertonic saline injections. This treatment consists in giving 300 c.c. of a 5 per cent solution of sodium chloride every other day. The rationale seems to be extremely vague. Our own investigations have shown that the following points are of importance: First, there results an increase in the circulating amount of fluid following the infusion of hypertonic saline; since 300 c.c. of a 5 per cent solution are given, the body has to provide another 1500 c.c. to make the solution isotonic. The total amount of the increased circulating fluids, therefore, will be in the neighbourhood of 1500–1800 c.c. Secondly, this increased amount of circulating fluid will increase the blood-pressure. It will also produce a considerable burden on the heart, and therefore if this treatment is being contemplated it is essential to assess the cardiac status. Thirdly, the increased amount of fluid is made up mainly, if not solely, by the tissue fluid and therefore a marked drop in the viscosity of the blood is registered. Lastly, the withdrawal of fluid from the tissue spaces reduces œdema, which is invariably present in advanced cases. Consequently, we are dealing with a larger amount of fluid with a lower viscosity under a higher pressure, with the result that a larger amount of blood percolates through the narrowed peripheral channels.

I have come to the end of my lecture. In giving this short survey I have tried to deal with some of the current concepts concerning the physiology and pathology of peripheral vascular disease, and to cover some of the more important points in diagnosis and treatment of these conditions. In doing so I have followed my own experience, gained in the laboratory and at the bedside, and have expressed our own convictions and what we have come to regard as the truth. "Truth", as Sir Heneage Ogilvie writes, "is of our discovery and not of our making." When we meet the truth we must meet it fearlessly for "when we see it face to face it may not be in the guise we imagined; it may override our most cherished theories, upset our hopes, even smash our careers". I have not hesitated to express my own views, which, as I fully realize, must be in contrast to the beliefs and fads held by some authors in this field and are contrary to some of the views expressed in some of the current textbooks.

I have dwelt to a greater extent on our own studies of the peripheral blood-flow largely in order to point the way to a more rational management of peripheral vascular diseases.

I am indebted to the South African Council for Scientific and Industrial Research and to the Marais Research Grant for grants covering expenses incurred during these investigations.

It is a great pleasure to acknowledge the assistance of my colleagues who entrusted their patients to my care.

To the Staff of the Trigonometrical Survey I am indebted for expert assistance in the construction of the portable plethysmograph.

Figs. 54, 57, 58, 65, and 69 in Part I of this paper are reproduced by courtesy of the *South African Medical Journal* and *Figs.* 62 and 68 by courtesy of the *American Heart Journal.*

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THE BLOOD-SUPPLY TO THE SIGMOID COLON AND RECTUM

WITH REFERENCE TO THE TECHNIQUE OF RECTAL RESECTION WITH RESTORATION OF CONTINUITY*

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In the performance of sphincter-saving resections of the rectum with restoration of continuity—operations which are now undergoing further trial in the treatment of rectal and rectosigmoid cancer—a major concern of the surgeon is to secure an adequate blood-supply to the portions of colon and rectum remaining for anastomosis. In this connexion an accurate knowledge of the anatomy and functional capabilities of the vessels supplying the distal colon and rectum is essential. Much has already been written on the subject by Sudeck (1908), Archibald (1908), Drummond (1914), Pope and Judd (1929), Steward and Rankin (1933), Sunderland (1942), and Bacon and Smith (1948), but in the writer's opinion there is still need for greater clarity on certain points, in particular the feasibility of combining really high ligation of the inferior mesenteric vessels with subsequent re-establishment of intestinal continuity. In this paper the anatomy of the arterial supply to the sigmoid colon and rectum has been re-investigated, with special reference to this question, in cadavers and in patients at operation.

THE ARRANGEMENT OF THE INFERIOR MESENTERIC ARTERY AND ITS BRANCHES

The distribution of the inferior mesenteric artery was examined post mortem in 75 adult cases in the Pathological Department of St. Mary's Hospital. The method employed was to divide the peritoneal adhesions on the outer side of the iliac colon as in the early stages of an abdomino-perineal excision and to mobilize the whole sigmoid loop and mesocolon by sweeping them medially so that they finally came to occupy a median anteroposterior plane. The arrangement of the vessels was then observed from the left side and the relationship of the bowel and the vessels to important landmarks, such as the bifurcation of the abdominal aorta, the promontory of the sacrum, and the peritoneal reflection, carefully noted. The colon and rectum from the level of the highest point on the left iliac crest to at least 2 in. below the peritoneal reflection off the front of the rectum were next removed and pinned out on a cork examination square in exactly the same manner as that in which they lay in the body after mobilization. The vessels were dissected sufficiently to make their course clear. The preparation was kept in a fresh condition throughout, fixation being avoided so as not to interfere with the natural length and elasticity of the bowel and vessels. Finally a drawing of the specimen was made, and, with the aid of the notes previously taken, the various anatomical landmarks in the environment from which it had been removed were inserted in the sketch.

Fig. 222 is a composite diagram showing the arrangement of the vessels and related parts found in 40 of the 75 specimens. It will be noted that the first sigmoid (S_1) and left colic (L.C.) branches arose conjointly, and that their common origin

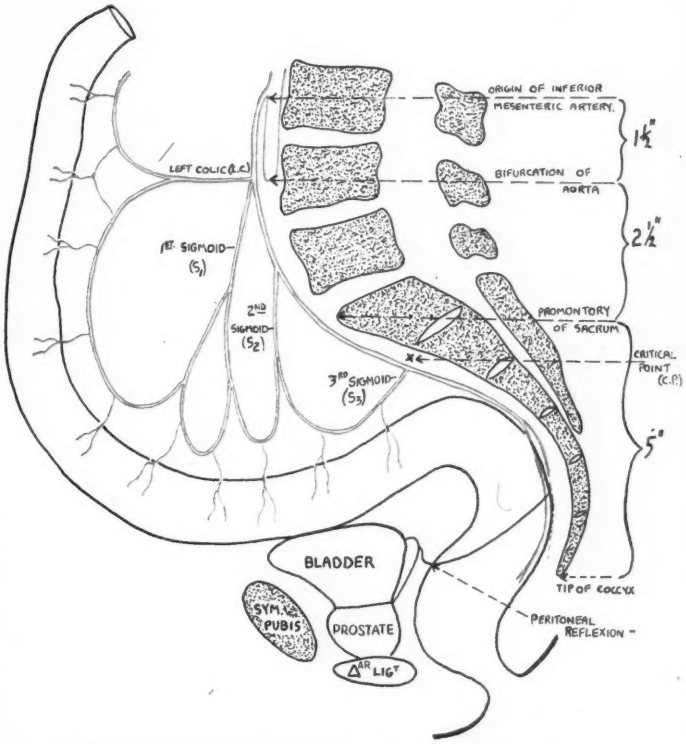

FIG. 222.—Diagram representing the commonest arrangement of the inferior mesenteric artery and its branches in 75 necropsy-room bodies. Special care has been taken to relate the arterial tree and bowel accurately to the bony pelvis and lumbar spine.

lay opposite the bifurcation of the abdominal aorta. In 5 specimens indeed the second sigmoid (S_2) also arose with the left colic as in *Fig.* 223, this disposition being most frequently observed when there were 5 or 6 sigmoid branches instead of the usual 3 or 4. In the remaining 30 specimens the vascular pattern corresponded more closely to the standard anatomy text-book description, the left colic and first sigmoid arteries being separated at their origin by a gap of $\frac{1}{2}$–$\frac{3}{4}$ in., as in *Fig.* 224.

THE PREPARATION OF THE SIGMOID STUMP IN RESECTION OPERATIONS

Fundamental Principles.—Because of the great predilection of carcinoma of the rectum to spread upwards along the lymphatics accompanying

* Being part of the substance of a thesis accepted for the degree of Ch.M. (Edin.) in June, 1948.

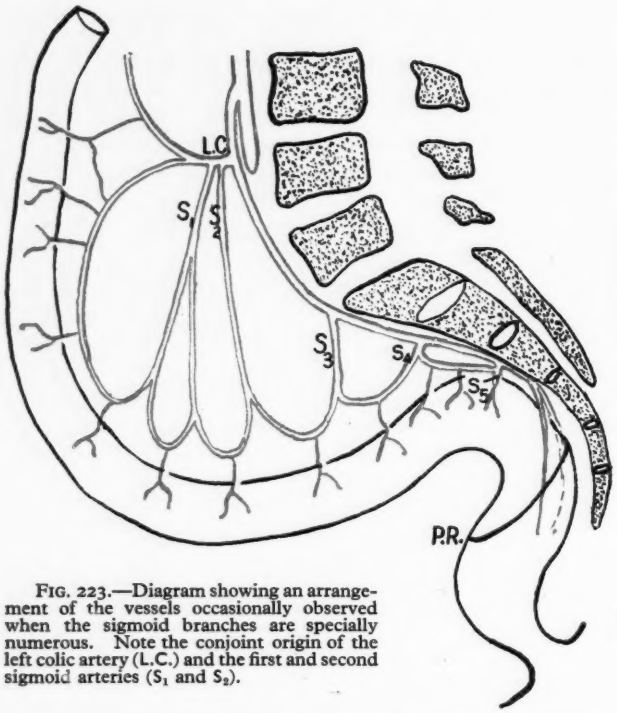

FIG. 223.—Diagram showing an arrangement of the vessels occasionally observed when the sigmoid branches are specially numerous. Note the conjoint origin of the left colic artery (L.C.) and the first and second sigmoid arteries (S_1 and S_2).

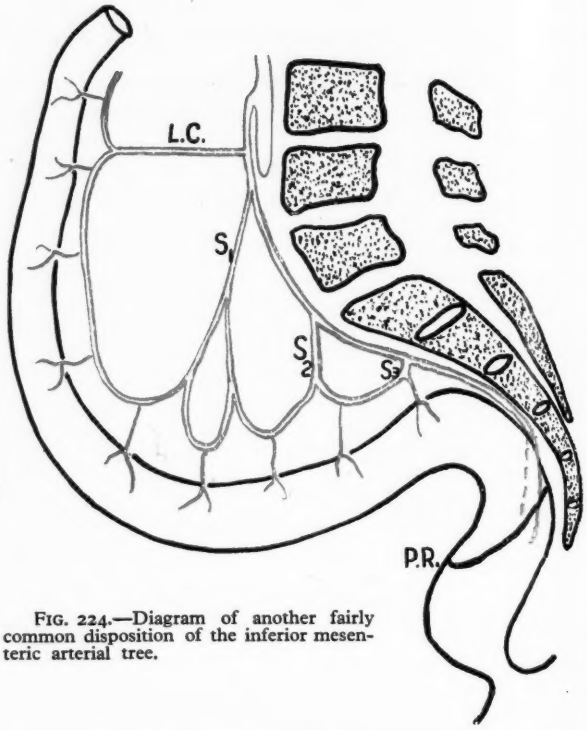

FIG. 224.—Diagram of another fairly common disposition of the inferior mesenteric arterial tree.

the superior hæmorrhoidal and inferior mesenteric vessels, it is important in a radical operation for this disease to tie these vessels as high as possible. In the classical combined abdomino-perineal excision most surgeons follow the advice of Miles (1926, 1939) and apply the main ligature roughly opposite the bifurcation of the abdominal aorta. As will be seen from *Fig.* 222, this will place it just below the conjoined origin of S_1 and L.C., or, if these two vessels arise separately, possibly between them. If resection operations are to compete with combined excision it is essential that in them also ligation should be carried out at this point and no lower, and rather than compromise on this issue, the attempt at resection should be abandoned in favour of a combined excision.

It is on this score that the sacral resection operations, previously so popular in Germany and Austria, must be condemned. Working through a sacral wound, the highest point at which the main vessels can be tied is usually just below the promontory of the sacrum, and often an inch or so lower, as in the old perineal excision of Lockhart-Mummery (Gabriel, 1945). References to *Fig.* 222 shows that this is $2\frac{1}{2}$ or $3\frac{1}{2}$ in. below the aortic bifurcation, which means that these sacral operations are much less radical than a combined excision.

The great advantage of incorporating an abdominal phase in the operation, as in the abdominal, abdomino-sacral, or abdomino-anal resections, is that it permits of ligation at a higher level and a proper display of the vessels to the sigmoid loop. Yet in most of the published accounts of the technique of these operations the site recommended for this ligature is not much above that chosen in sacral resections. Thus Wangansteen (1945) states that it should be applied opposite the promontory of the sacrum; Bacon (1945) places it at the level of the common iliac bifurcation, thus preserving virtually all the sigmoid vessels; and Muir (1948) ligates either between or below the sigmoid branches. These relatively low ligations are presumably inspired by the desire to preserve as much of the sigmoid circulation as possible in the hope of making available an extra long piece of well-vascularized colon for anastomosis. They are of course open to the serious pathological objection that they make the operation considerably less thorough than a combined excision, and, as far as subsequent restoration of continuity is concerned, as will be shown later, they are quite unnecessary and even disadvantageous. Study of our arterial preparations demonstrates that *ligation of the inferior mesenteric vessels may be accomplished exactly as in a combined excision and yet sufficient sigmoid colon preserved for re-establishing continuity*. This can be done in three ways.—

Method 1.—In the average case with a reasonable length of sigmoid colon, the most dependent point on the loop, which is some distance from its lower end, lies on the bladder, as in *Fig.* 222, and is almost on a level with the peritoneal reflection off the front of the rectum. Generally this piece of colon is supplied by S_1, so that by tying the inferior mesenteric vessels just below the origin of this branch and dividing the mesocolon parallel to it, as shown in *Fig.* 225, a sigmoid stump is obtained, with a direct blood-supply to its tip which can extend practically to the peritoneal reflection, and will therefore usually suffice for anastomosis after resection down to this level. For some at an even lower level it will also be adequate, because as Wangensteen (1945) has emphasized, when the rectum is mobilized so that it assumes a straight course upwards from the anus instead of lying in the concavity of the sacrum, the level of the peritoneal reflection may rise as much as 2 in. (*Fig.* 226). If a longer sigmoid stump is

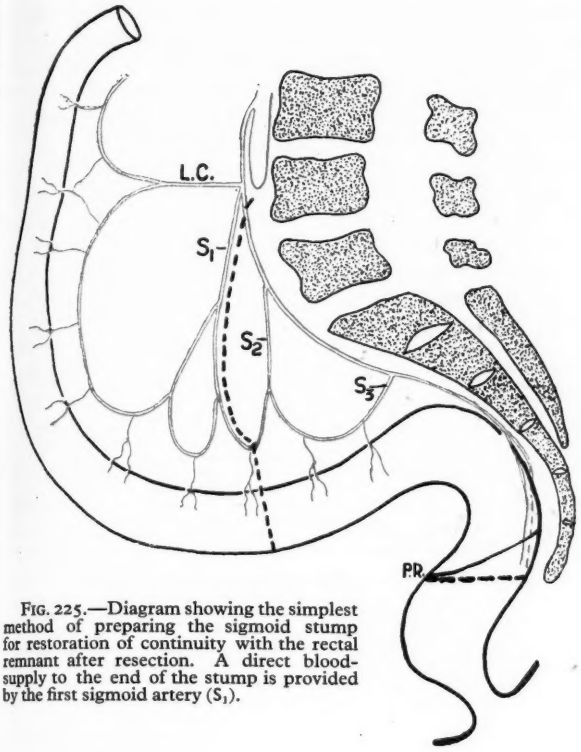

FIG. 225.—Diagram showing the simplest method of preparing the sigmoid stump for restoration of continuity with the rectal remnant after resection. A direct blood-supply to the end of the stump is provided by the first sigmoid artery (S_1).

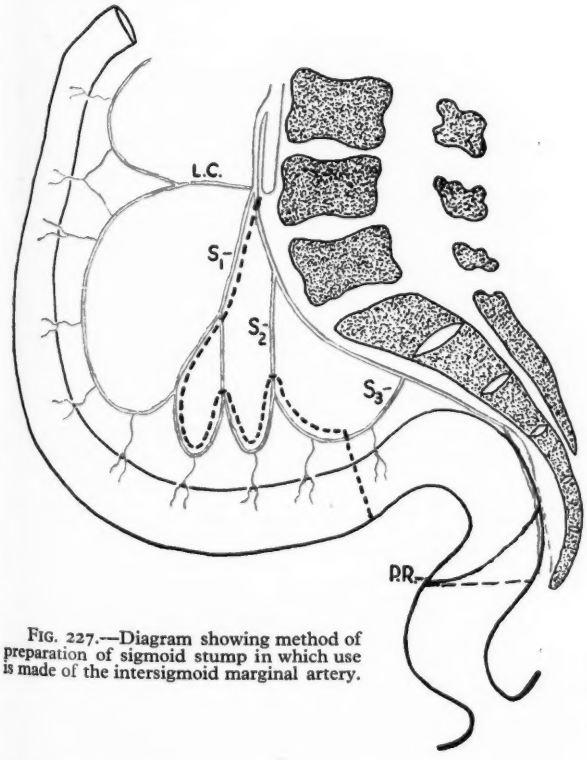

FIG. 227.—Diagram showing method of preparation of sigmoid stump in which use is made of the intersigmoid marginal artery.

required it may be secured by either of the succeeding two methods.

Method 2.—In this method the inferior mesenteric artery and mesocolon are divided as in *Fig.* 223, but, instead of sectioning the bowel as there shown, it and the intersigmoid arcades for some distance below this are preserved intact, as in *Fig.* 227. The length obtained by this manœuvre depends on the width of the gaps between the sigmoid arteries, which is actually very variable, but generally 2–3 in. of lengthening can be accomplished in this way.

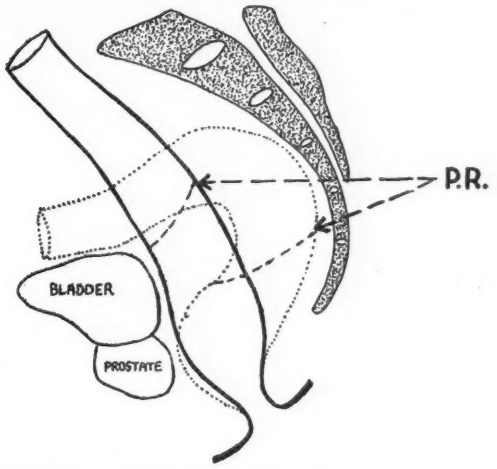

FIG. 226.—Diagram to show how the peritoneal reflection rises when the rectum is mobilized and lifted out of the hollow of the sacrum.

Method 3.—Here after ligation of the inferior mesenteric vessels just below the common origin of S_1 and L.C., S_1 itself is divided close to its commencement as in *Fig.* 228 (or in the cases where it and L.C. arise independently, as in *Fig.* 224, the main inferior mesenteric ligature is applied between these two branches). Thus reliance is placed entirely on the descending branch of L.C. and the marginal artery between it and S_1 for the supply of the stump. This opening up of the large 'window' between S_1 and L.C. usually secures an additional 2–3 in. of

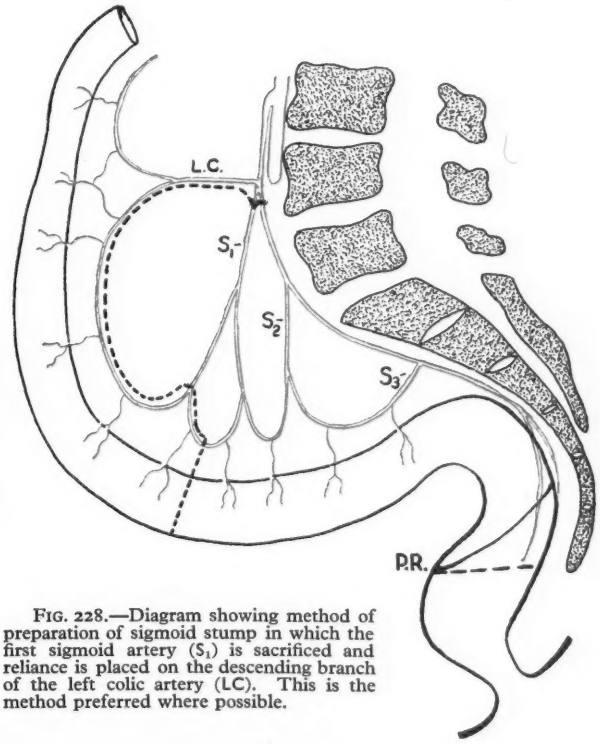

FIG. 228.—Diagram showing method of preparation of sigmoid stump in which the first sigmoid artery (S_1) is sacrificed and reliance is placed on the descending branch of the left colic artery (LC). This is the method preferred where possible.

colon, and further permits of another inch or two being obtained if necessary by division of the peritoneum at the outer side of the descending colon and mobilization as far as the splenic flexure. Until S_1 has been divided, freeing of the descending colon in this fashion is futile, because the factor determining the extent to which the end of the stump can be brought down is the intact S_1.

Experimentation with the arterial specimens showed that however short the sigmoid loop might be, enough colon could always be provided by one of these manœuvres, most often Method 3, to permit of *end-to-end union* with the anorectal remnant after resection 1 in. or so below the anterior peritoneal reflection. With ligation farther down on the inferior mesenteric trunk opposite the sacral promontory it was found that though a redundancy of colon often existed above, the extent to which its lower end could be drawn down into the pelvis was very much restricted by the intact second or third sigmoid arteries, so that the effective length of stump was very much less. In an *abdomino-anal or 'pull-through'* type of resection, in which the colon is not sutured to the upper end of the rectal stump but is brought through it so as to project 2 in. or so from the anus or to be sutured to the anal verge, a specially long piece of colon is necessary, at least 4 or 5 in. longer than for an ordinary low intra-abdominal anastomosis. By utilizing both Methods 2 and 3 for lengthening the sigmoid stump it was found possible to obtain this quantity of bowel in 41, or approximately 53 per cent, of our post-mortem specimens; in the remainder the length of colon secured fell short of that required and in these cases abdomino-anal anastomosis would have been impossible.

CONFIRMATORY OBSERVATIONS AT OPERATION

Steward and Rankin (1933) have rightly stressed the fallacy of assuming that, because a good arterial supply can be demonstrated in post-mortem preparations, the circulation will in fact be adequate in the living subject. Many of the patients with carcinoma of the rectum are elderly and suffering from advanced arterial disease, so that the possession of fair-sized colic vessels is no guarantee that they will transmit blood in an efficient manner. Observations such as have been recorded above cannot therefore be expected to carry much weight unless supported by evidence from actual patients. Further information has thus been sought from cases during operation for rectal excision.

In 36 patients sigmoid stumps were prepared by the Methods 1, 2, and 3 already described. The circulation in the affected portion of colon was then carefully observed. It was found that the colour of the bowel afforded an extremely reliable indication as to viability; if it remained pale pink and did not turn blue, one could be absolutely certain that the blood-supply was adequate. Absence of visible pulsation in the vessels of the colon wall was a much less trustworthy criterion; first, because it was often very difficult to be sure whether the vessels were pulsating or not; and, secondly, because even when pulsation was absent enough blood might still reach the bowel to keep it alive. In 4 of our cases, for example, after ligation prolonged scrutiny failed to reveal any pulsation, but the colon remained a good colour and section of one of the small intramural vessels produced brisk arterial hæmorrhage. It was presumed, therefore, to be viable, and was utilized for anastomosis with the rectal stump; the suture line healed perfectly in all 4 cases and there was no necrosis. The fluorescein test described by Hatfield, Buyers, and Walkling (1945) for determining the viability of gut was also tried in a few instances, but was not found to possess any advantages over these simpler methods.

In 17 cases the preparation of the colon was according to Method 3, relying on the descending branch of the left colic artery. The sigmoid stump thus secured was viable in all, as indicated by its colour and the occurrence of bleeding from its cut end or from an experimentally divided vessel in its wall. In 12 of these cases the operation was completed as a restorative resection, and the subsequent uneventful healing of the anastomoses in these patients with no evidence of necrosis showed that our confidence in the adequacy of the circulation had not been misplaced.

In 8 cases Method 2, preserving the first sigmoid and the intersigmoid marginal artery, was chosen, and viable stumps were obtained in all. Four were employed for restoring continuity subsequently, and their suture lines healed without incident.

In 9 cases the sigmoid stump was prepared as in Method 1, the first sigmoid artery supplying the end of it; again the blood-supply was deemed sufficient in all. Five proceeded to restorative resection, and of these 4 had perfect union, but in one some necrosis took place leading to a breakdown of the posterior half of the suture line, which healed later.

Finally in 3 cases, where a particularly long piece of colon was required for an abdomino-anal anastomosis, recourse was had to a combination of Methods 2 and 3, the stump being supplied by the descending branch of the left colic and beyond this by the intersigmoid marginal artery. The colon thus prepared seemed quite viable at operation, but post-operatively necrosis took place in 2 cases, in one involving fully 4 in. of bowel—both cases, however, surviving the mishap. It would seem that these long stumps are particularly prone to thrombosis of their vessels, due either to the operative trauma or to later sepsis.

USE OF TRANSVERSE COLON OR AN ILEAL LOOP TO RESTORE CONTINUITY

Very occasionally it will be found impossible to provide a satisfactory sigmoid stump for anastomosis after resection because of the existence of pathological conditions such as double or treble carcinomata, polyposis intestini, or very extensive diverticulosis, in all of which the greater part of the sigmoid colon may have to be sacrificed. In some of these cases resection with restoration of continuity might still be achieved by adopting the method recommended by Kümmell (1899) and Archibald (1908) of dividing the left colic artery at its origin and mobilizing the descending colon thoroughly so

as to enable it, supplied solely by the middle colic through the marginal artery, to be drawn down into the pelvis. They adduced evidence by injection experiments, observations in animals, and a few clinical trials that the circulation would be adequate under these circumstances. A safer procedure is probably that of Treves (1898), which consists of resecting up to the splenic flexure and then swinging down the left end of the transverse colon nourished by the long left branch of the middle colic artery. Lloyd-Davies (1948) and the writer have recently made use of the transverse colon in this way, with satisfactory results. A third possibility is to utilize a loop of ileum, isolated except for its blood-supply, as a graft to bridge the gap between the stump of iliac colon and the rectum, as was done by Fenwick (1914), Stone (1928), and Wangensteen (1943).

For the performance of either of these latter two manœuvres, however, the transverse colon or lower ileal coils must be particularly dependent. It may be of interest to record that it was found possible to fashion a transverse colon stump capable of extending down to the peritoneal reflection in 46 of the 75 necropsy-room bodies examined, and in 36 of these it reached as far as the anus. In approximately half of the bodies also it appeared that one of the lower ileal loops might have been of value for grafting purposes after rectal or rectosigmoid resection.

THE BLOOD-SUPPLY TO THE ANO-RECTAL STUMP

Drummond (1913) and Steward and Rankin (1933) have emphasized that the main artery to the rectum is the superior hæmorrhoidal and that the middle and inferior hæmorrhoidal vessels are of very much less importance and anastomose little with the terminals of this vessel. It might be imagined therefore that when the supply from above is cut off as in performing a resection, the contribution from the middle and inferior hæmorrhoidals would prove inadequate to sustain the remaining portion of rectum and anus, especially if a long rectal stump were left. Steward and Rankin (1933) indeed considered necrosis inevitable under such circumstances, and recommended that whenever division of the superior hæmorrhoidal (or inferior mesenteric) artery was necessary the whole rectum should be excised.

Practical experience with 21 abdominal resection has shown the fallacy of this doctrine. Even when a large rectal remnant is preserved at operation its cut upper edge usually bleeds sufficiently to dispel any doubt as to the adequacy of its blood-supply. It should be emphasized that this is so even when the lateral ligaments with their contained middle hæmorrhoidal vessels have been divided. After operation, moreover, there has never been any necrosis of the rectal stump even when this was of considerable length and had been freed down to the anorectal ring. Clearly the blood-supply to the rectum from below must be much more abundant than has hitherto been thought. It would appear to be derived from the inferior hæmorrhoidal vessels, and perhaps from other vessels in the vicinity of the rectum, as postulated by Pope and Judd (1929). During the perineal phase of a complete excision of the rectum it is striking how constantly and freely the inferior hæmorrhoidal arteries bleed when cut; it is also hard to believe that the numerous vessels, which are divided and cause such sharp hæmorrhage when the pubo-rectales and pubo-coccygei muscles are severed at the side of the rectal ampulla, do not contribute to the supply of the rectum itself.

SUMMARY

An examination of the arrangement of the blood-vessels to the rectum and distal colon in 75 necropsy-room bodies, supplemented by observations on 36 patients at operation, has established the following points:—

1. If a resection operation is to be as radical as a combined excision it is essential that the ligature on the inferior mesenteric vessels should be placed not lower than the level of the bifurcation of the abdominal aorta.

2. With the main ligature so sited, it is possible in practically every case to prepare a sufficiently long stump of sigmoid colon with an adequate blood-supply to permit of *end-to-end union* with the rectal stump after resection. This piece of colon is nourished by the first sigmoid artery alone or in combination with the intersigmoid marginal artery, or by the descending branch of the left colic artery.

3. When restoration of continuity by an *abdomino-anal or pull-through* technique is contemplated a very much longer colon stump is required; this can be provided in only approximately half the cases. Long stumps of this type seem specially liable to post-operative thrombosis of their vessels.

4. When the entire sigmoid colon has to be sacrificed, as for example in certain cases of double carcinomata, it may be possible to resect up to the splenic flexure or middle of the transverse colon and anastomose the remainder of the transverse colon to the rectal stump, or to use an ileal graft to bridge the gap between iliac colon and rectum.

5. The blood-supply to the anorectal stump after resection, in which the superior and often the middle hæmorrhoidal vessels have been divided is surprisingly abundant. It would appear to be derived from not only the inferior hæmorrhoidal arteries, but also numerous unnamed branches in the anterior parts of the levator muscles.

I wish to express my indebtedness to Professor W. D. Newcomb for permission to investigate the anatomy of the blood-vessels to the colon and rectum in the necropsy-room material of his department at St. Mary's Hospital; and to Mr. H. J. R. Bussey, B.Sc., for preparing most of the diagrams.

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PRIMARY CARCINOMA OF PENILE URETHRA

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THIS case is reported because of the rarity of carcinoma of the urethra.

A careful survey of the literature in 1938 by H. A. R. Kreutzmann and B. Collof (1938) revealed only 150 cases.

The present writer has been able to trace 6 more cases of primary carcinoma in the male urethra (R. F. Hickey and R. C. Coleman, 1944; K. Ikeda, F. E. B. Foley, and J. Roseman, 1943; W. N. Wishard jun., 1943; C. C. Herger and H. R. Sauer, 1942; V. M. Rau, 1942; H. H. Young, 1939).

This case is also of interest because it shows how carcinoma of the urethra mimics stricture of the urethra and its complications. This mimicry is apt to lead to delay in diagnosis as is stated in several previous reports (F. R. O'Neil, 1921; E. M. Watson, 1929; C. B. Huggins and G. M. Curtis, 1929). In many recorded cases the first indication of malignancy has been the finding of a necrotic or gelatinous mass on incising a urethral swelling instead of finding pus as was expected (Young, 1926), and this occurred in the case recorded below. In the first stage a urethral stricture is simulated; when a tumour appears associated with obstruction, a periurethral abscess is suggested; and later, when fistulæ are added, the appearance is that of suppurative periurethritis with fistulæ. E. D. McCrea (1940) and O'Neil (1921) point out that most cases have required two operations before a diagnosis was established—the second one including a biopsy.

Pain in carcinoma of the urethra is frequently severe—it occurs spontaneously and is increased during micturition. Free bleeding may occur without obvious cause and these two signs, pain and bleeding per urethram with a penile swelling, are suggestive of carcinoma in the urethra. A swelling in the urethra due to a gumma is less painful and tender and should be excluded by a Wassermann reaction.

The relation of carcinoma of the urethra to chronic urethritis and stricture also merits attention. At least half of the recorded cases were stated to have had gonorrhœa or a stricture for an antecedent (W. Rizzi, 1921; H. A. R. Kreutzmann and B. Collof, 1939; R. F. O'Neil, 1921). Although stricture and chronic urethritis are common complaints, the frequency of these or a similar pathological process such as leucoplakia or fistulous tracts seem to be too commonly recorded as precursory lesions for them to be merely coincidental. They must be one aetiological factor amongst many others predisposing to the condition.

It has been suggested that instrumentation for the treatment of stricture increased the predisposition to carcinoma. J. J. Robb (1928) records one case where bougies were passed 111 times in five years. He postulates that carcinoma of the urethra was due to this. In the case recorded below the patient had a stricture present for ten years and attended fairly regularly for bouginage until one year before admission.

PATHOLOGY

Carcinomata of the urethra are most commonly squamous-celled, much more rarely papillary or transitional-celled. The squamous type may arise from metaplasia of the urethral mucosa, which is mostly columnar (from the membranous part to the fossa navicularis). In many cases metastases occur late in the course of the disease.

Metastases spread through the lymphatics to the deep inguinal glands and on to the external iliac and hypogastric glands. Although some of these cells pass there via the deep subinguinal glands, others go in lymphatics which run with the vessels to the bulb and internal pudic vessesls and so pass directly to the intrapelvic glands (Rouvière, 1932; Wood Jones, 1946).

From this it can be seen that absence of enlargement of the glands in the groin does not mean that metastatic spread has not occurred. It has also been pointed out by numerous authors that enlargement of the inguinal glands may be due to sepsis and not to metastatic deposits (E. D. McCrea, 1940). This also suggests that any block dissection limited to the glands of the groin may be incomplete and that a block dissection should theoretically include the glands along the external iliac and hypogastric arteries. I cannot find any record of this having been done.

Besides lymphatic spread, metastasis occurs in the corpora cavernosa, as may be seen in the photograph of a section of the urethra in this case. (*See Fig.* 230.)

TREATMENT

Deep X-ray therapy, radium, and radical and partial surgical removal of the urethra are all recorded in the literature as treatment for carcinoma of the urethra.

Unfortunately in most cases the late diagnosis makes irradiation ineffective, although in early cases it would seem ideal if combined with a suprapubic cystotomy (E. M. Wilson, 1929). In all cases a preliminary cystotomy is valuable as a means of

reducing sepsis by preventing urine from contaminating the operation area. Most cases of carcinomata of the penile urethra when treated surgically have a partial or total removal of the penis and usually a block dissection of the glands in the groin. Carcinoma of the bulbous part is rarely diagnosed early enough for a radical operation, although Young (1939) has described an ingenious resection of the bulbous urethra with reunion of the remaining parts.

In cases where the radical operation is performed, it seems better, from physiological reasons as well as for anatomical convenience, to remove the testicles and scrotum (J. Everidge, 1943; G. Grey Turner, 1925), although some American authors (Kreutzmann and Collof, 1939; Huggins and Curtis, 1929) do not advise this.

The extirpation of the inguinal glands is a question of some nicety.

As was pointed out above, the inguinal glands are only on one metastatic route. Their enlargement may be due to sepsis rather than metastasis, and the absence of enlargement does not mean that metastasis has not occurred. It seems reasonable, therefore, to perform a block dissection at the time of the radical operation only if the inguinal glands are palpably enlarged if sepsis has been reasonably controlled. The radical operation is one of some severity, as severe bleeding may occur when separating the crura from the pubic rami. If sepsis is marked and the patient's condition poor, it is probably wiser to wait a week or more for the block dissection of the groins. If then the glands remain consistently small, deep X-ray therapy may be more suitable than operative treatment. If the glands remain enlarged, a block dissection should be carried out.

PROGNOSIS

Prognosis is better for carcinoma in the anterior urethra than in the posterior urethra, as would be expected from its more obvious and accessible position. Kreutzmann and Collof (1939) give a 56 per cent recovery rate for carcinoma of the penile urethra and only 14·3 per cent recovery for carcinoma proximal to the penile urethra.

CASE HISTORY

O. B., aged 55 years, an intelligent, active man—a general labourer.

A urethral stricture was known to be present for ten years. In 1938 he was an in-patient in Withington Hospital and had his stricture dilated under anæsthesia. Since then he attended approximately at monthly to three-monthly intervals until 1947, when he left the district for one year.

March 5, 1948: He attended a surgical Out-Patient Clinic complaining of dysuria: a slowness of micturition, burning after micturition, and an interruption of the stream at the end of micturition.

ON EXAMINATION.—Temperature 97·4°, pulse 96, blood-pressure 125/85. Chest, somewhat barrel-shaped, resonant, equal, fair movements, normal breath-sounds, few crepitations at bases. Abdomen, no masses palpable, bladder not distended, no penile abnormality or inguinal adenitis noted. Urine, normal. No blood or albumin.

He was instructed to re-attend the Out-Patient Clinic for bouginage.

March 17: He complained of a painful swelling in his penis. He was found to have a tender swelling in the middle third of the ventral aspect of the penis and a purulent urethral discharge. T. 97·6°, P. 100. The swelling was not reddened. His admission was arranged on the diagnosis of a periurethral abscess, but was delayed 24 hours to suit patient's domestic arrangements.

March 20: *Incision of penile swelling.* No pus but thick gelatinous material found. Indwelling catheter inserted. After the second day patient passed urine through his penile wound.

March 31: *A suprapubic cystotomy and biopsy was* performed in view of the exuberance of the penile swelling, the urethral sinus, and urinary sepsis.

Blood-urea estimation: Blood-urea 44 mg./100 c.c.

Blood-count: R.B.C. 4,720,000; C.I. 0·9; Hb 12·4; W.B.C. 15,000.

April 6; W. R. negative.

April 8: Biopsy report: "Section of tissue shows wide infiltration with sheets of epithelium of the transitional-cell type. Mitotic figures are fairly numerous and the appearances are those of an actively growing urethral carcinoma."

The patient was referred to the Christie Hospital for radium treatment. Dr. Tod and Dr. M. J. Gibson found the condition too advanced for irradiation.

The clinical appearance of the lesion was that shown in the coloured illustration (*Fig.* 229).

FIG. 229.—Carcinoma of the urethra. Painting of the condition seen at operation.

April 19: *Radical removal of penis and scrotum* through scrotal encircling incision (Pearce Gould, 1882). The inguinal glands were not removed. Catheter sutured into urethra. Free bleeding occurred when the crura were detached from the pubic rami. The dissection was done with a diathermy point.

PATHOLOGICAL DESCRIPTION OF SPECIMEN.—

1. Section shows a fungating tumour of the penis consisting of a mass of carcinoma cells of the transitional-cell type. The growth is heavily infected, polymorphonuclear leucocytes are numerous, and some areas necrotic and hæmorrhagic. A transitional-cell carcinoma of the urethra (*see Fig.* 231).

2. Sections of penis proximal to the fungating ulcer show widespread infiltration of the corpora with masses

of carcinomatous tissue of the transitional-cell type, and many of the blood-sinuses are filled with growth. The carcinoma retains its transitional-cell structure throughout, but in a few areas assumes a more spindle-cell type and mitotic figures are numerous (*see Fig.* 231).

POST-OPERATIVE TREATMENT.—Blood transfusion given—4 pints and 2 pints plasma. Penicillin 20,000 units 3-hourly. Sulphacetamide 1·0 g. q.h.

April 22: Gradual improvement.

April 23: Tab. Ferrous Sulph. gr. 6. and vitamin C 200 mg. given t.d.s. Oblique incision healing well, some

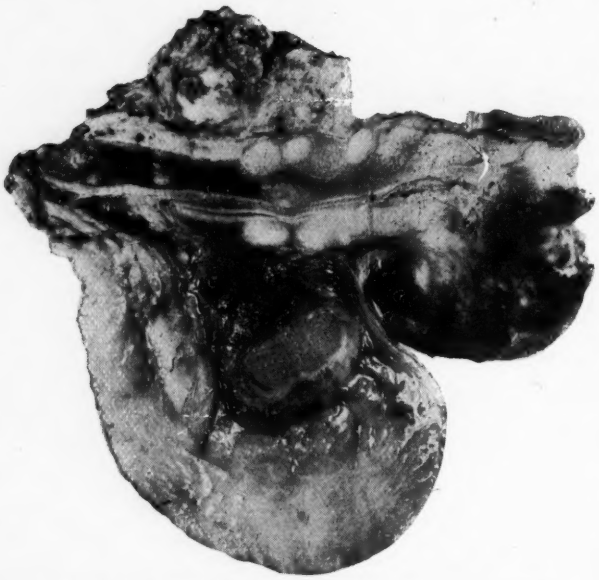

FIG. 230.—Carcinoma of the urethra shown in section. Note the extensive carcinoma fungating through the penis, with masses of carcinoma in the corpora cavernosa.

sloughing of the skin edges in the median wound. Eusol dressings applied.

May 2: At 8 p.m. sudden hæmorrhage from the median wound adjacent to the left side of urethra—controlled by a firm pack; 6–8 oz. of blood lost. Two pints of blood given intravenously—renewed hæmorrhage occurred during the transfusion, but this, again, was controlled by packing.

May 5: General improvement, no further hæmorrhage. Hb 5·65 g. (42 per cent). Two pints of blood given intravenously.

May 10: Hb 6·6 g. (48 per cent). Median wound still septic. A culture from it yielded a heavy growth of *B. proteus*, diphtheroids, and non-hæmolytic streptococci. *B. proteus* was insensitive to penicillin; the diphtheroids and cocci were sensitive to penicillin. The median wound was dressed with 2·2 per cent aq. sol. phenoytol b.d.

May 14: Wound cleaner, patient ambulant—getting about with urethral catheter at first clamped off and later removed.

June 13: General improvement. Patient is passing urine through his urethra normally and has full control of his urine. The inguinal gland on the right side became increased in size and did not appear to be subsiding with the decrease in wound sepsis. It was, therefore, decided to do a block dissection of the glands in the right groin. Left groin—glands not enlarged clinically. Hb 69 per cent.

June 17: *Block dissection of right groin* under spinal anæsthesia. Right oblique inguinal incision. A quadrate mass of superficial fascia and lymphatic glands was dissected off the femoral vessels in the groin and a small corrugated drain inserted. Three postage-stamp skin-grafts were applied to the median wound.

HISTOLOGY OF INGUINAL GLANDS AND GRANULATION TISSUE.—

1. Inguinal glands: Hyperplasia of lymph-follicles. Dilatation of sinusoids. No evidence of secondary deposits of growth.

2. Granulation tissue from median wounds shows infiltration with polymorphonuclear leucocytes, but no evidence of new growth.

POST-OPERATIVE TREATMENT.—

June 22: Penicillin 40,000 units three hourly. General improvement, wounds healing well, fair take of median graft.

July 1: Discharged to Out-Patient Department.

July 14: General condition good, wounds almost fully healed, small granulation in median wound cauterized. Attending weekly for dilatation of urethra.

Aug. 4: Patient has had jaundice for three days, urine dark, containing bile. No enlargement of liver. ? Homologous serum jaundice. Urine contained a few pus cells, hyaline and granular casts. Culture yielded *B. proteus*.

Aug. 6: Serum-bilirubin 8·5 mg./100 ml. Alkaline phosphatase 35·8 units. Thymol turbidity 9 units. Colloidal gold 2 units. Liver enlarged two fingers below costal margin.

Aug. 18: Still jaundiced, liver similar. Serum-bilirubin 9·7 mg./100 ml. Alkaline phosphatase 28 units/100 ml. Thymol turbidity 12 units. Colloidal gold 5 units.

Aug. 25: Jaundice decreasing. Liver still two fingers below costal margin. Serum-bilirubin 2·6 mg. 100 ml. Alkaline phosphatase 17 units. Thymol turbidity 8 units. Colloidal gold 4 units. General condition improving.

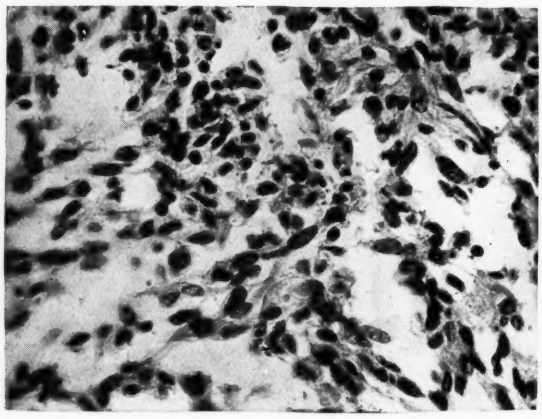

FIG. 231.—Photomicrograph of section of tumour showing transitional-celled epidermoid carcinoma of urethra. (× 200.)

Sept. 8: General condition improved—jaundice almost gone from skin, but still present in conjunctivæ. Serum-bilirubin 1·8 mg./100 ml. Alkaline phosphatase 18 units/100 ml. Thymol turbidity 8 units/100 ml. Colloidal gold 5 units/100 ml.

Sept. 22: General improvement. Jaundice disappeared. Serum-bilirubin 0·9 mg/100 ml. Alkaline phosphatase 15 units/100 ml. Thymol turbidity 7 units/100 ml. Colloidal gold 4 units/100 ml.

Sept. 27: In-patient for two days. Bouginage—spinal anæsthesia. 11/14 bougie passed easily. Mild stricture of meatus.

Oct. 6: No. 12 gum-elastic bougie passed easily. Patient feels well and is expecting to begin work. He is to attend for routine urethral dilatation, there being a tendency to form a stricture at the external orifice. Serum-bilirubin 0·3 mg./100 ml. Alkaline phosphatase

17 units/100 ml. Thymol turbidity 4 units/100 ml. Colloidal gold 2 units/100 ml.

Oct. 18: Returned to work.

The above liver-function tests indicate recovery from toxic liver damage, due probably to the serum given post-operatively, i.e., to homologous serum jaundice.

SUMMARY

1. This case history demonstrates that rapidly spreading carcinoma of the urethra can be eradicated surgically with success when too far advanced for irradiation.

2. It is suggested that, ideally, any glandular removal for metastases whould include the iliac glands as well as the inguinal glands.

3. Preliminary suprapubic cystostomy is of value in reducing wound sepsis.

4. Operative treatment removing the primary neoplasm is probably worth while in all but the most advanced cases of carcinoma of the anterior urethra.

The author is greatly indebted to Mr. H. T. Cox, whose case this was, for much encouragement and for permission to operate on his patient; to Dr. Stent, for numerous pathological investigations and reports; and to Dr. Greenwood, Medical Superintendent, and Mr. J. B. Macalpine, for much assistance.

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POSTGASTRECTOMY SYNDROMES

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SUBTOTAL gastrectomy has become the operation of choice for the surgical treatment of severe peptic ulceration, particularly in young people with high gastric acidity. Unfortunately digestive disturbances following this operation are very common and patients with these disturbances are seen in increasing numbers in hospital out-patient departments. The symptoms of which they complain have been investigated, but no agreement has yet been reached as to their cause, frequency, and treatment. Publications dealing with a few cases of the more dramatic types of postgastrectomy syndrome give the reader an erroneous impression of their incidence and importance. Confusion has also arisen about the nomenclature of the syndromes, and it is suggested that this important point should receive more consideration in subsequent publications on this subject.

PREVIOUS WORK

There is no agreement about the frequency of digestive complaints following subtotal gastrectomy. American writers accept an average figure of 15 per cent (Jordan, Miller, Custer, and others). In Edinburgh Gilbert and Dunlop found an incidence of approximately 35 per cent after 45 consecutive gastrectomies. The incidence varies in each published series, depending doubtless upon the keenness of the follow-up investigator and the criteria adopted, as well as upon the type of operation performed. There is no doubt, however, that digestive symptoms follow subtotal gastrectomy sufficiently frequently to constitute a major problem in the post-operative management of these patients.

Descriptions of postgastrectomy syndromes have also differed in clinical details. Within the last few years three have attracted special attention in British, American, and Scandinavian literature:—

1. Early postprandial or dumping syndrome.
2. Late postprandial or hypoglycæmic syndrome.
3. Failure to gain weight.

The symptoms of the *dumping syndrome* have given rise to the greatest controversy. Some writers, mainly American, regard the syndrome as principally vasomotor in character. The following description is typical (Eusterman, 1947): "In this vasomotor syndrome, distress usually begins ten to 14 days after operation; it may date from the first solid food taken post-operatively. The syndrome is painless but disabling. Symptoms begin suddenly during or ten minutes after eating and consist of profound nausea and weakness, a generalized unpleasant sense of warmth, cold diaphoresis of the face (especially the forehead), and cardiac palpitation. There may be hypermotility of the small bowel with excessive borborygmus ending in explosive diarrhœa. The attack, usually lasting ten to sixty minutes, may be prevented or relieved by the patient's assuming a supine position."

It will be seen that little stress is laid on upper abdominal discomfort, bilious vomiting, and regurgitation, etc., which are described as characteristic of the dumping syndrome by other workers (Irvine;

Adlersberg and Hammerschlag; and others). One patient described by Gilbert and Dunlop did not develop symptoms until one hour after a meal but was, notwithstanding, classified as an example of the dumping syndrome. It is not surprising, therefore, that different causes have been ascribed to this syndrome by different workers.

Glaessner (1940) was of the opinion that it depended on the rapid rise of blood-sugar to hyperglycæmic levels known to follow carbohydrate ingestion in gastrectomized patients. Zollinger and Hoerr (1947) also found that hyperglycæmia accompanied early postprandial symptoms following gastrectomy. Experimentally, however, rapid raising of the blood-sugar level in man by intravenous or intrajejunal glucose administration does not produce symptoms, and, moreover the syndrome is often apparent before a significant rise in blood-sugar has occurred. Adlersberg and Hammerschlag (1947) studied 14 patients with persistent digestive symptoms following gastrectomy, and they attributed the early postprandial symptoms—fullness after meals, nausea, eructations, dizziness, fatigue, weakness, palpitation, sweating, and headache to mechanical factors, namely, jejunal distension due to precipitate emptying of the gastric remnant. The general features of the syndrome—sweating, weakness, palpitation, dizziness, faintness, etc.—were attributed to reflex vagotonia secondary to jejunal distension. Gilbert and Dunlop, however, have pointed out that these symptoms are not characteristic of vagal stimulation and are not relieved by atropine in full doses. Furthermore, similar symptoms have been reported in gastrectomized patients following vagotomy (Moore, Chapman, Schulz, and Jones, 1947), and it has been shown that the stomach is able to appreciate sensory stimuli after complete division of the vagus nerves (Andrus, 1947; Dragstedt, Harper, Tovee, and Woodward, 1947). This led Irvine (1948) to suggest that jejunal distension produced symptoms by stimulation of the *sympathetic* nervous system, and he also considered that early postprandial symptoms were mechanical in origin, since symptoms occurred after the heaviest meal of the day and were unrelated to carbohydrate intake.

In 1948 Gilbert and Dunlop claimed that early postprandial symptoms following gastrectomy were invariably hypoglycæmic in origin. They based their conclusion on the study of 17 patients and showed that glucose-tolerance curves in these patients differed significantly from those of 28 symptom-free gastrectomized patients. The curves of the former group followed closely the lag-storage curves of Maclean, and minimum blood-sugar levels were considerably lower than in the symptom-free patients. They believed that there was a greater tendency to hypoglycæmia in patients experiencing symptoms and reported 2 patients in full who exhibited severe hypoglycæmic attacks approximately one hour after meals. Lawrence (1936) and Evensen (1942) have recorded lag curves in patients after short-circuiting gastric operations, and the latter author has described frequent examples of alimentary hypoglycæmia following subtotal gastrectomy. There is thus no unanimity of opinion regarding the cause of early postprandial symptoms in gastrectomized patients.

It is generally agreed that *late* postprandial symptoms are hypoglycæmic in origin. The clinical features—weakness, sweating, palpitation, tremor, headache, and on some occasions loss of consciousness, are consistent with this diagnosis and authentic accounts of severe hypoglycæmic attacks occurring several hours after meals are recorded by Barnes (1947), Evensen (1942), Zollinger and Hoerr (1947), and many others. There is, however, still no agreement as to the mechanism of production of the alimentary hypoglycæmia in these patients. It is known that glucose tolerance curves following gastrectomy are frequently abnormal, indicating deranged carbohydrate metabolism. The name 'lag-storage' has been applied by Maclean to this type of curve, and its principal features are as follows: The blood-sugar in the fasting patient usually reveals no abnormality, but after the ingestion of 50 g. of glucose it rises rapidly to very high levels and glycosuria often occurs. Blood-sugar levels of as high as 300–350 mg. per cent have been recorded during this initial part of the test. Thereafter a very rapid fall occurs and hypoglycæmic levels are frequently reached at the one- to two-hour readings, accompanied by classical symptoms of this condition. The blood-sugar then gradually rises to approximately fasting levels. This curve differs from the normal in the initial rapid rise to hyperglycæmic levels and the equally rapid fall to hypoglycæmic levels which quickly follows it. It is now accepted that the early rise is due to very rapid absorption of glucose from the upper bowel, for the following reasons:—

1. Lag-storage curve is known to occur most frequently in patients with rapidly emptying stomachs, e.g., patients with duodenal ulcer, after gastroenterostomy and subtotal gastrectomy (Lawrence, 1936).

2. Wohrle (1936), Evensen (1942), and Lawrence (1936) were able to reproduce the hyperglycæmic phase of the curve in healthy people by intubation and introduction of glucose into the duodenum. Lawrence preferred the name 'oxyhyperglycæmic curve' to that of 'lag-storage curve'. None of these workers, however, were able to induce blood-sugar curves showing a significant hypoglycæmic phase following this procedure. Kalk and Meyer (1932) demonstrated this phase, however, in blood-sugar curves following intrajejunal intubation and glucose administration, and it is noteworthy that it occurs not infrequently in normal people and may be of sufficient degree to provoke hypoglycæmic symptoms.

Other explanations have been advanced from time to time for this rapid initial rise in blood-sugar following carbohydrate ingestion in certain people. Maclean regarded it as being due to delay in the liberation of insulin by the pancreas, and from this hypothesis the name 'lag-storage curve' was derived. Hetényi and Pagány (1928) suggested that it was due to excessive breakdown of liver glycogen during carbohydrate absorption, but this view is untenable since galactose-tolerance curves exhibit the same abnormality (Barnes, 1947) and galactose is not reformed from liver glycogen. The first phase in a blood-sugar curve of this type, therefore, is most satisfactorily explained on the grounds of

unusually rapid absorption of sugar from the intestine secondary to rapid emptying of the stomach.

The subsequent rapid fall in the blood-sugar to hypoglycæmic levels, however, has provided the main field for argument. Since this is the phase of the curve which is accompanied by symptoms, it is important to have a clear conception of its aetiology. In the first place it is not due to lack of available carbohydrate in the alimentary canal, since Malmros, Evensen, and others have shown that alimentary hypoglycæmia can occur in patients exhibiting this curve while a quantity of glucose still remains in the stomach. Four possible explanations remain :—

1. It may be due to an excessive insulin response by the pancreas to the initial hyperglycæmia—a reactive hypoglycæmia. Many workers adopt this simple explanation—Evensen, (1942) ; Gilbert and Dunlop, (1947). Barnes (1947), however, raises certain objections to its unreserved acceptance. He points out that hypoglycæmia seldom follows hyperglycæmia induced by intravenous glucose (Lennox and Bellinger, 1927 ; Hartman and Foster, 1932 ; Ross, 1938), and that hypoglycæmic symptoms after subtotal gastrectomy are often delayed for several months—a fact which is difficult to explain if this hypothesis is correct.

2. It may be due to the development of insulin sensitivity. It is well known that high carbohydrate diets increase insulin sensitivity while low carbohydrate diets decrease it (Himsworth, 1939). It is possible that postprandial hyperglycæmia in gastrectomized patients may react in a similar way to a high carbohydrate diet in a normal subject. Barnes believes that this is the true explanation of hypoglycæmic symptoms following gastrectomy, and has demonstrated increased insulin sensitivity in at least one typical case.

3. It may be due to an excessive demand for sugar on the part of the liver and other tissues containing glycogen. This explanation is untenable since capillary-venous blood-sugar differences are normal (Gilbert and Dunlop, 1947).

4. It may be due to failure of the adrenaline response of the body to the rapidly falling blood-sugar. That this hypothesis is incorrect is suggested by the fact that hypoglycæmic syndromes developing in gastrectomized patients are associated with ample clinical evidence of adrenaline activity—dilated pupils, tachycardia, pallor, etc. (Barnes, 1947).

Further work is clearly required on this aspect of the problem before a full explanation of the facts is given.

The third postgastrectomy syndrome, namely *loss of weight* after operation, has been widely recognized but has received remarkably little investigation. Wollaeger, Mandred, Weir, and Osterberg (1946) showed that gastrectomized patients lost more fat, total solids, and nitrogen in the fæces than normal subjects. The differences were very small, but were considered to be significant enough to cause gradual loss of weight if allowed to continue indefinitely.

Adlersberg and Hammerschlag, on the other hand, were unable to increase the weight of their patients by giving a high-calorie diet supplemented by vitamin concentrates and liver extracts. They regarded the loss of weight as psychological in many cases. Whatever be the cause of this syndrome there is no doubt that it is regarded with anxiety by the patient, who often considers it a clear indication that the operation has not been entirely successful.

The relationship of these symptoms to the type of gastrectomy performed has not received the attention it deserves. Gilbert and Dunlop found no significant difference in their incidence in patients who were treated with the retrocolic Polya anastomosis and in those who had the Hoffmeister valved type. In Barnes's series, however, the latter type of anastomosis was uniformly successful in preventing subsequent symptoms in over a hundred consecutive gastrectomies. Mimpriss and Birt (1948) found that, in general, antecolic anastomosis gave less satisfactory results than short-loop retrocolic anastomosis. The length of the afferent loop in the former operation was thought to be the cause of postcibal distress in these patients. They also found that anastomosis of the afferent loop to the greater curve led to a high incidence of proximal-loop filling and have abandoned this operation.

Treatment of these syndromes has depended on the particular viewpoint taken by the clinician regarding the aetiology. Thus Gilbert and Dunlop used high-fat diets, prescribed as six small meals in the day, with the intention of preventing hypoglycæmia. Alternatively, 1 oz. of olive oil and ½ gr. of ephedrine were given before meals. Barnes also used high-fat low-carbohydrate diets with the intention of decreasing insulin sensitivity in his patients and claimed good results. Adlersberg and Hammerschlag drew attention to the psychological element in the syndromes and stressed the importance of dealing with it adequately. An accurate assessment of treatment is difficult, as all are agreed that improvement occurs spontaneously in the majority of cases.

PERSONAL OBSERVATIONS

The investigations to be described were made on 124 patients, 102 males and 22 females, who had undergone subtotal gastrectomy for a variety of gastric ailments. Professor C. F. Illingworth performed 92 operations in the Western Infirmary, Glasgow, and has kindly provided the following brief description of his technique : " Distally, duodenum divided approximately 1 in. beyond pylorus and end enfolded. Proximally, the stomach divided on lesser curvature close below cardia (in order to do this, as a routine the left gastric artery is divided before it reaches the stomach and its œsophageal branch is also divided separately). On the greater curvature the stomach is divided above the lowest of the vasa brevia, and sometimes at a higher level still. Jejunum brought in front of transverse colon and joined to cut end of stomach, with proximal end of jejunum to lesser curvature. Proximal loop of jejunum about 6 in. in length. Size of stoma equal to full width of stomach at point of section."

Mr. W. J. M. Brandon performed the remainder at Law Hospital, Lanarkshire. His technique is similar except that a retrocolic type of anastomosis

is preferred. The conditions for which gastrectomy was performed were as follows :—

Chronic duodenal ulcer	73
Chronic gastric ulcer	17
Gastric ulcer and duodenal ulcer	9
Stomal ulcer following gastro-enterostomy	7
Early carcinoma of stomach	17
Fibrosis of stomach following corrosive poisoning	1
	124

Patients were seen at intervals which varied from a few weeks to six years after gastrectomy, the average interval being eleven months. No patient was included in the series who suffered from stomal ulceration or post-operative gastritis. Blood-sugar estimations were performed by the method of Hagedorn and Jensen, and capillary blood was used throughout.

1. Glucose-tolerance Tests in Gastrectomized Patients.—A standard glucose-tolerance test was performed on 86 patients. Normal results were obtained in 52, i.e., the fasting blood-sugar was between 80 and 120 mg. per cent, the maximum rise after 50 g. of glucose did not exceed 180 mg. per cent, a fall to approximately fasting levels occurred within two hours, and glycosuria was absent during the test. Mild hypoglycæmic symptoms occurred in four patients in this group at the periods of minimum blood-sugar levels. The remaining 34 patients exhibited a glucose-tolerance curve of the lag type. *Table I* summarizes the results in the latter

Table I.—Glucose-tolerance Tests in Gastrectomized Patients

Case No.	Time in Hours					
	F.	½	1	1½	2	2½
	mg. per cent					
1	94	174	286	142	74	80
2	104	241	161	68	94	105
3	71	136	195	148	110	70
4	116	204	200	78	64	82
5	81	292	94	53	71	104
6	92	143	206	74	88	91
7	104	293	100	61	60	85
8	74	119	283	171	68	104
9	85	182	180	102	94	88
10	96	148	231	84	59	109
11	93	174	200	62	60	79
12	88	185	317	106	51	82
13	106	172	234	182	91	116
14	70	195	191	102	81	96
15	93	246	85	78	104	86
16	85	148	209	79	74	96
17	72	172	186	194	72	106
18	96	159	214	131	100	104
19	104	207	338	104	72	78
20	116	258	117	61	66	94
21	108	166	194	57	83	96
22	81	205	106	63	68	74
23	76	161	244	86	82	88
24	68	185	74	74	95	92
25	85	241	314	116	96	81
26	110	311	308	171	126	70
27	94	265	188	84	60	78
28	110	268	142	60	70	94
29	86	242	161	61	74	94
30	74	181	186	92	63	90
31	100	199	288	141	75	75
32	96	264	136	93	111	108
33	92	200	195	105	78	110
34	85	294	191	117	56	75

group. Glycosuria was present during the test in every case and 21 patients experienced hypoglycæmic symptoms at the period of minimum blood-sugar levels.

2. The Aetiology of the Lag Curve in Gastrectomized Patients.—It is now generally agreed that the first or hyperglycæmic phase of the curve is due to rapid absorption of glucose from the bowel following precipitate emptying of the gastric remnant. The second or hypoglycæmic phase is attributed by some to the development of insulin sensitivity (Barnes) and by others (Maclean) to an exaggerated insulin response by the pancreas to the early hyperglycæmia. To elucidate this problem the following investigations were carried out :—

a. An attempt was made to discover how soon after subtotal gastrectomy a lag-curve response to the ingestion of 50 g. of glucose could develop. A glucose-tolerance curve was performed on each of 6 patients a few days prior to gastrectomy and thereafter at five-day intervals for several weeks. Two patients, originally showing normal responses, exhibited typical lag-curve responses five days and ten days after operation respectively, and each showed a well-marked hypoglycæmic phase with characteristic symptoms. The remaining four still showed normal curves many months after operation.

b. Twelve patients were selected who exhibited a lag-curve response to the ingestion of 50 g. of glucose and who developed hypoglycæmic symptoms during the test. An insulin sensitivity test was performed on each, employing the test used by Barnes, i.e., 0·05 unit per kg. of body-weight of soluble insulin was injected intravenously into the fasting patient and blood-sugar was estimated at four-minute intervals (Fraser, Albright, and Smith, 1941). Only one patient showed increased insulin sensitivity with blood-sugar readings as follows :—

Time in minutes	F.	4	8	12	16	20	24
Blood-sugar mg. per cent	92	92	61	48	38	39	46

It is therefore unlikely that the hypoglycæmic phase of a lag-storage curve depends on the development of insulin sensitivity, since the phase may be well marked a few days after gastrectomy before there is time for this to become significant, and insulin sensitivity tests in patients exhibiting the curve are normal in the great majority of cases.

3. Digestive and Metabolic Abnormalities following Subtotal Gastrectomy.—It is a fact that only 9 gastrectomized patients out of a total of 124 were completely free from digestive symptoms following operation. Furthermore, patients with stomal ulceration or post-operative gastritis (Christiansen, 1942) were excluded from this series, which therefore represents the successful gastrectomy. In the great majority symptoms were mild, transitory, and detracted little from the value of the operation. In approximately 10 per cent, however, they were severe enough to prolong convalescence and prevent return to work for an average period of five months. These figures emphasize the seriousness of the problem.

In the author's own investigations postgastrectomy symptoms fell into six groups, in the following order of frequency and importance: (1) The dumping syndrome; (2) Failure to gain weight after operation; (3) Psychoneurotic manifestations;

(4) Bilious regurgitation and vomiting; (5) Hypoglycæmic symptoms; (6) Deficiency syndromes.

Many patients experienced more than one of the syndromes and were able to give an indication of the relative severity of each. The syndromes were arranged in this order, the patient's account of his symptoms taking precedence over all other findings, biochemical and otherwise.

1. THE DUMPING SYNDROME

Incidence.—Ninety patients (75 per cent) of the present series suffered at some time from this syndrome. It was the most common and was the principal factor in prolonging postoperative convalescence. It occurred at all ages and in both sexes with equal frequency, and did not appear to be related to the duration of symptoms prior to operation. or to the nature of the original gastric lesion, although the impression was gained that it was never so severe in patients who had undergone gastrectomy for gastric ulcer or early carcinoma of the stomach. Its onset dated from a few weeks after operation in 90 per cent of cases. It was not delayed longer than three months in any patient, and in those patients in whom this delay occurred the syndrome was mild and only appeared after unusually heavy meals.

Clinical Features.—The symptoms varied greatly in severity, but were remarkably uniform in type. The main feature was a feeling of upper abdominal distension coming on shortly after meals. This was almost invariably accompanied by a feeling of drowsiness and lassitude, which in some cases was severe enough to force the patient to sit or lie down. Nausea occurred in the majority of cases, with vomiting in approximately 50 per cent. The type of vomiting which occurred in this syndrome was very characteristic. No matter how soon after meals it occurred it consisted of bile unmixed with food. Patients were seen who vomited several pints of pure bile fifteen minutes after a moderate-sized meal. These symptoms—upper abdominal distension, lassitude, nausea, and bilious vomiting—were the most constant. Other symptoms, however, were well marked in many patients. A cold sweat, flushing of the head and face, flatulence, palpitations, and intestinal borborygmi were often present, and transient syncopal attacks occurred in 2 patients. An explosive attack of watery diarrhœa accompanied by the passage of much flatus occurred in 6 patients.

The syndrome came on at an interval which varied from a few minutes to thirty minutes after a meal. Its onset often forced the patient to stop eating and, it is stressed, was never delayed longer than half an hour after a meal. The average time of onset in this series was approximately ten minutes. Its duration varied from twenty mintues to two and a half hours, with an average of approximately one hour.

It is interesting to consider the principal types of meal which induced symptoms. As Irvine (1948) has pointed out, the bulk of the meal was the important factor in this respect. Many patients avoided symptoms completely by eating only small meals. Milk puddings, soups, salads, potatoes, and heavy prepared dishes invariably caused a severe attack and usually the patient avoided them. There were some patients, however, who maintained that a particular type of food precipitated an attack, e.g., meat, cheese, beer, etc. Each time this statement was encountered a careful inquiry always revealed the fact that the meals containing the suspected food were also heavier than the others. There was one type of food, however, which often precipitated attacks and could not be dismissed as a specific cause on these grounds, viz., very hot, and to a lesser extent very cold, food. It was found that some patients invariably exhibited a dumping syndrome when even small quantities of very hot or very cold food was taken, e.g., a cup of hot tea or a glass of cold water. Moreover, the dumping syndrome induced was atypical in that upper abdominal distension and bilious vomiting were usually inconspicuous, the general features of the syndrome predominating—hot flushes, sweating, and lassitude.

Of the 90 patients who suffered from the dumping syndrome, 15 were unable to resume work for an average period of five months following operation. These patients had severe symptoms, as may be appreciated from the following case records :—

Case 1.—F. B., aged 33, labourer.

Subtotal gastrectomy performed for chronic duodenal ulcer Feb. 21, 1945. During the three weeks he was in hospital he had no gastric symptoms. On returning to his home the following symptoms developed: Fifteen minutes following a meal he experienced a tight feeling across the upper abdomen and became nauseated. Within ten minutes thereafter severe bilious vomiting occurred, which gave immediate relief. If vomiting did not occur spontaneously it was often induced for this reason. These symptoms were accompanied by a feeling of intense weakness and drowsiness and he was forced to lie down when they appeared, this, too, giving relief. Sweating, palpitations, and giddiness also occurred, but were not usually conspicuous. Symptoms have gradually improved during the last three years, but still occur after a heavy meal.

Case 2.—J. M., aged 40, works manager.

Subtotal gastrectomy April 13, 1948, for chronic duodenal ulcer. Since operation is unable to eat a normal meal. If he does so he experiences epigastric fullness, nausea, and bilious vomiting ten to fifteen minutes later and lasting for an hour. Drowsiness and tiredness are also prominent, and he is often forced to lie down after meals. Head flushing, flatulence, and borborygmi are also apparent, but sweating is unusual. There has been little improvement in the last few months.

Case 3.—W. N., aged 32, miner.

Subtotal gastrectomy for chronic duodenal ulcer Jan. 15, 1947. Two days before discharge from hospital he developed bilious vomiting fifteen minutes after meals, associated with tightness in the upper abdomen, faintness, flatulence, sweating, and occasionally diarrhœa. These symptoms were relieved by vomiting and by lying down. X-ray examination of the stomach and gastroscopy were performed because of the severity of the bilious vomiting, but nothing abnormal was found. He was treated with frequent small meals and has gradually improved, but still has similar symptoms if he does not lie down for half an hour after meals.

Twenty-eight patients had symptoms of moderate severity, seldom interfering with work but constituting a considerable handicap. The following case record is typical of this group :—

Case 4.—L. R., aged 47, dock labourer.

Subtotal gastrectomy for prepyloric ulcer April 23, 1947. Immediately after operation he developed

epigastric fullness and bilious vomiting half an hour after meals. Lassitude and borborygmi became apparent, rapidly followed by an attack of diarrhœa. These symptoms quickly improved and disappeared completely in six months. He has at present no gastric complaints except for a little epigastric fullness and sweating fifteen minutes after an unusually heavy meal.

The remaining 47 patients had very mild symptoms of the above order, which occurred only after unusually heavy meals or rapid drinking of hot or cold liquids.

The total duration of the syndrome averaged fourteen months. It sometimes disappeared within three months, but not infrequently persisted for years. It showed a tendency to improve gradually, but severe examples were seen five or six years after operation. This syndrome is probably identical with the dumping syndrome of American literature (Mix, 1922), which, however, stresses the vasomotor phenomena rather than epigastric fullness and bilious vomiting.

Aetiology.—In order to come to more definite conclusions on the cause of this syndrome the following investigations were made :—

1. Blood-sugar estimations were made at ten-minute intervals during a typical attack in eleven patients. *Table II* summarizes the results. No patient showed hypoglycæmia in any phase of the syndrome.

Table II.—Blood-sugar Levels taken at Ten-minute Intervals throughout Typical Attacks of the Dumping Syndrome

Case	Blood-sugar in Milligrammes Per Cent										
1	138	146	114	128	138	116	144				
2	94	128	162	166	144	118	86	94			
3	114	126	134	128	126	122					
4	143	128	116	94	97						
5	88	117	126	138	144	161	174	161	142	128	
6	123	136	148	161	152	148	158	141	128	126	115
7	73	116	121	81							
8	84	96	112	118	124	128	126	114	108		
9	79	148	178	146	112	78					
10	141	161	161	128	94	114	112	101	98		
11	142	158	151	112	94	114					

2. Three meals of different type were prepared and each was given to six patients with classical symptoms of the dumping syndrome. The contents of the meals were as follows :—

Meal A :—

		CHO	*Protein*	*Fat*
Mashed turnip	80 g.	2	—	—
Cabbage	50 g.	1	—	—
Poached egg	1	—	6	6
Bacon	60 g.	—	8	27
Butter (in vegetables)	5 g.	—	—	5
Banana	80 g.	16	—	—
Glucose (mixed with banana)	30 g.	30	—	—
		49	14	38

Meal B: Identical with above except that 110 g. of stewed apple sweetened with saccharin (4 g. CHO) was substituted for the 110 g. banana and glucose dish.

Meal C: Identical with *A* except that cabbage and mashed turnip were omitted and the butter was added to the bacon.

Meals *A* and *B*, therefore, were identical in weight and bulk and differed only in carbohydrate content, *A* containing ten times that of *B*. Meal *C* had approximately half the weight and bulk of *A* and *B*, and had the same carbohydrate, protein, and fat content as *A*.

The symptoms which followed the meals in the 6 patients were compared as regards time of onset, character, severity, and duration. Blood-sugar estimations were started ten minutes after a meal had been eaten and continued at ten-minute intervals throughout the period of symptoms. Each patient was encouraged to eat the meal in twenty minutes to avoid variations in rates of distension of the gastric remnant. *Table III* summarizes the results of the investigation.

It is clear from the table that weight and bulk are the principal factors contributing to the production of the dumping syndrome, the carbohydrate content of the meal having little effect. It is also worthy of note that again no hypoglycæmic blood-sugar readings were obtained during the period of symptoms.

3. A Ryle's stomach tube with a balloon attached was passed into the upper jejunum under X-ray control in 4 patients with typical dumping syndromes. Inflation of the balloon with air reproduced the syndrome, including its vasomotor components, in 2 patients, induced a mild sensation of fullness in the epigastrium in 1, and failed to produce symptoms in 1 patient. This finding will be discussed later.

Treatment.—Ephedrine ½ gr. before meals, tincture of belladonna pushed to the limits of tolerance, and olive oil ½ oz. before meals were all tried with little success. Mild and moderate cases were considerably relieved by a short rest after meals, or, alternatively, by substituting six small meals for three larger ones during the day. Severe cases, however, persisted in spite of this treatment, although the majority were relieved to some extent. Two further methods of treatment are worthy of consideration. A few patients were so severely affected that they became a social and economic burden. Three patients of this type were met with in the present series. One man was forced to remain at home for several hours following even a moderate-sized meal. Bilious vomiting, intense epigastric fullness, and exhaustion occurred, lasting for as long as two hours. Gastroscopy and X-ray examination were persistently negative. Somewhat reluctantly, and as a last

resort, he was given a stomach tube, instructed in its use, and told to aspirate the stomach contents an hour or so after meals. Aspiration proved to be unnecessary, as the act of leaning forward with the tube in position invariably resulted in as much as two pints of pure bile flowing from the stomach, rapidly relieving his symptoms. He started to eat larger meals without discomfort, gained weight, and

Table III.—Showing the Severity and Duration of the Dumping Syndrome following Meals of Varying Weight and Carbohydrate Content

Meal *A* —Heavy meal; high carbohydrate content.
Meal *B* —Heavy meal; low carbohydrate content.
Meal *C* —Light meal; high carbohydrate content.
The time of onset and termination (in minutes) of each symptom after the meal are indicated in the respective columns.

Cases	Epigastric Fullness	Nausea	Vomiting	Lassitude	Vasomotor Upset	Blood-sugar in Milligrammes per cent at 10-min. Intervals following the Meal
1. *A*	Severe, 5–45	Severe, 10–45	Severe, 45	Severe, 5–70	Severe, 15–40	128 145 166 148 138 148 114
B	Severe, 0–30	Severe, 10–30	Severe, 30	Severe, 15–60	Severe, 20–40	116 121 108 114 123 106 92
C	Moderate, 15–80	Mild, 30–70	—	Moderate, 40–60	—	147 156 174 156 144 122 123 138
2. *A*	Severe, 0–60	Severe, 0–60	Moderate, 60	Moderate, 10–70	Mild, 10–20	148 176 192 188 142 158 134
B	Severe, 0–70	Severe, 0–70	Moderate, 70	Severe, 15–60	Mild, 10–15	98 114 116 122 126 188 129
C	Mild, 10–15	—	—	—	—	118 195
3. *A*	Severe, 0–90	Severe, 0–80	—	Mild, 15–60	Severe, 20–70	123 144 156 142 154 136 138 152 131
B	Severe, 10–80	Severe, 5–40	—	Moderate, 10–60	Severe, 20–45	102 117 112 98 116 128 126 119
C	Moderate, 10–30	Mild, 0–25	—	—	—	138 166 151
4. *A*	Moderate, 0–45	Mild, 15–45	Moderate, 45	Moderate, 5–50	—	138 161 158 147 131
B	Mild, 10–50	Moderate, 25–50	Moderate, 50	Moderate, 15–40	—	116 124 118 100
C	—	—	—	—	—	165 128 142 161
5. *A*	Severe, 10–30	Severe, 10–30	Severe, 30	—	Moderate, 15–40	164 142 124 155
B	Severe, 0–20	Severe, 5–20	Severe, 20	—	—	96 98 104 114 102
C	Moderate, 5–40	Moderate, 10–40	Moderate, 40	—	Moderate, 10–15	154 126 161 162
6. *A*	Moderate, 5–80	Moderate, 20–60	—	Moderate, 10–80	—	128 142 156 161 147 138 142 151
B	Severe, 0–60	Mild, 10–20	—	Moderate, 15–70	—	116 124 108 122 131 114 128
C	—	—	—	—	—	161 108 172 146 114

returned to work within six weeks. At the present time he carries out this procedure only very occasionally and is practically free from symptoms. The other two patients who were similarly affected were cured by the operation of anastomosing the afferent and efferent loops of bowel. In one of these men a diagnosis of obstruction of the afferent loop was possible on clinical grounds, as may be appreciated from his case history.

Case 5.—J. P., aged 43, farmer.

Gastro-enterostomy performed in 1928 for duodenal ulcer. Six months later subtotal gastrectomy was performed for stomal ulceration. Thereafter, for five years, he suffered from a severe dumping syndrome which gradually disappeared. He remained free from gastric symptoms until March, 1948, when the following symptoms developed. After a heavy meal, particularly one containing soup or milk pudding, a tight feeling developed across the epigastrium. If this was ignored and eating continued, it increased and spread into the throat and chest and down both arms. Palpitation, faintness, and sweating became evident, culminating in explosive bilious vomiting which relieved his symptoms immediately. A radiograph showed barium passing normally from the gastric remnant into the jejunum and gastroscopy revealed no abnormality. The nature of his attacks suggested an obstructive condition and a laparotomy was performed in May, 1948. The afferent loop was found to be unduly long and marked kinking was noted at a point a few inches from its junction with the stomach. An entero-anastomosis was performed between the proximal and distal loops. He has been free from symptoms since this operation.

A second patient had subtotal gastrectomy performed in 1945 for stomal ulceration. For one year he was completely free from symptoms. He then developed epigastric fullness, faintness, nausea, and severe bilious vomiting after meals which did not respond to rest, small meals, or the administration of atropine. These symptoms persisted for two years. Repeated X-ray examinations, test-meals, and gastroscopies did not reveal any abnormalities. In August, 1948, laparotomy was performed and

a long dilated proximal loop found with no clear evidence of obstruction at any point. An entero-anastomosis was performed as before and the patient has since remained free from symptoms. A third patient has recently been treated in a similar fashion by Professor Illingworth with excellent results (personal communication).

2. FAILURE TO GAIN WEIGHT AFTER SUBTOTAL GASTRECTOMY

This was second only to the dumping syndrome in nullifying the good effects of subtotal gastrectomy for peptic ulcer. Its importance was greatly exaggerated by the majority of patients, who regarded it as clear evidence that operation had been a failure. In the present series an accurate weight prior to operation was available in 86 patients. This figure excluded those who had lost a considerable amount of weight before operation as a direct result of their gastric lesion. The results of weighing each patient six months after operation were as follows :—

Exceeded pre-operative weight	7
Attained pre-operative weight or within a few pounds of it	44
½ to 1 st. below pre-operative weight	16
1 to 1½ st. below pre-operative weight	8
1½ to 2 st. below pre-operative weight	7
2 to 2½ st. below pre-operative weight	4
	86

A loss of weight of over half a stone therefore occurred in approximately 40 per cent of patients, while considerably less than 10 per cent gained weight as a result of operation.

The following investigations were carried out in the 35 patients who were underweight (Group A) and the 51 who had attained pre-operative weight (Group B) :—

1. The incidence and severity of other digestive abnormalities in the two groups were compared. Each patient of Group A exhibited these symptoms and in over 80 per cent they were classed as severe or moderately severe. In Group B their incidence was also over 80 per cent, but only 30 per cent were classified as severe or moderately severe.

2. The dietetic records of the patients in each group were compared. No accurate comparison was possible since these records depended for their accuracy on the patient's own story, no standard diet being prescribed in any patient following operation. It was found that the diet of the patients of Group A was on the average distinctly smaller in quantity than that taken by those of Group B. The severity of concomitant digestive symptoms in the former group, particularly epigastric fullness after meals, was the main reason for this difference. Patients exhibiting this symptom in a severe form invariably ate smaller meals, avoiding fatty meals, milk puddings, and other foods of high calorie value.

3. Supervision of the diet in patients of Group A, particularly as regards quantity, resulted in a gradual gain in weight in the majority, although it must be admitted that this was very slow, averaging no more than one to two pounds a month.

4. Seven patients of Group A had, however, no obvious reason for remaining underweight. Their diet was more than adequate, no vomiting or diarrhœa was present, and, apart from failure to gain weight and a little epigastric fullness after unusually heavy meals, they were in excellent health. A fat-tolerance test was performed on these patients. Each was kept on a standard diet containing 50 g. of fat per day for a week. The total fat loss per day in the fæces was estimated during the last four days. The fæcal fat was found to average 12 to 20 per cent of the dried-stool weight in 5 patients and to be under 5 per cent in 2.

Five underweight patients therefore out of a total of seven who had no obvious reason for failing to gain weight had definite steatorrhœa. This inevitably contributed towards failure to reach pre-operative weight and was almost certainly an indication of more widespread absorptive failure of protein and carbohydrate (Wollaeger, 1946). Further investigation of this important abnormality in gastrectomized patients is obviously required.

3. NEUROTIC SYMPTOMS FOLLOWING GASTRECTOMY

The great majority of the patients in the present series had at one time suffered from peptic ulcer. The psychological factor in this condition has been stressed on numerous occasions, many regarding it as a psychosomatic illness. It is not surprising, therefore, that a high incidence of neurotic syndromes was encountered. Three of these occurred with sufficient frequency to merit further discussion, particularly as each was commonly misdiagnosed.

1. Syncopal Attacks.—Six patients described the following syndrome : At a period which bore no relationship to the taking of meals a sudden feeling of weakness and giddiness was experienced. No loss of consciousness occurred, but the patient was forced to sit or lie down during an attack. Pallor, sweating, nausea, and palpitation occurred. The whole attack lasted a few minutes only and rapidly disappeared without specific treatment. A hypoglycæmic origin for the symptoms was at first suspected, particularly as exercise was a common precipitating factor. This diagnosis was discarded, however, because of the following facts :—

a. Blood-sugar estimations made during several attacks showed no level below 96 mg. per cent.

b. Intravenous glucose and subcutaneous adrenaline had no alleviating effect.

c. The attacks were too brief and were as likely to occur half an hour after a carbohydrate meal as at any other time.

d. Each patient exhibiting them had marked anxiety symptoms of a different order, e.g., chronic fatigue, phobias, feelings of apprehension, frequency of micturition, etc.

2. Neurotic Vomiting.—This occurred in ten patients. The vomit was bilious in character, occurred at no fixed time after meals, was often misdiagnosed as part of a dumping syndrome, but differed from it in the following points :—

a. Frequently occurred several hours after food.

b. Was unaccompanied by other manifestations of the dumping syndrome, for example, epigastric fullness, sweating, lassitude, etc.

c. When it occurred soon after a meal the size of the meal was of no importance. It was as severe after eating half a slice of toast as after a three-course lunch.

d. In each case it served a useful function to the patient and was accompanied by other neurotic symptoms. Usually it was employed as an excuse to change to a more congenial occupation, or avoid family responsibilities.

3. Neurotic Air Swallowing.—Five patients were able to bring on an attack of upper abdominal discomfort associated with bilious regurgitation and vomiting closely resembling the dumping syndrome, by swallowing air. Flatulence was a common symptom of the dumping syndrome but not of the severity of that occurring in these patients, who stated that relief was *only* experienced after enormous quantities of gas had been brought up, and that the size of the preceding meal was not related to the severity of symptoms. The process of aerography was watched in several patients, and it was only after it continued for some time that epigastric fullness was experienced and bilious vomiting occurred, the latter invariably accompanying the explosions of gas from the stomach and occurring at no other time. Each patient exhibiting this syndrome had strong psychoneurotic tendencies and was cured of the attacks by receiving a simple explanation of their cause and prevention. It is a remarkable fact that two of these patients were so severely affected that at the height of an attack manifestations of tetany occasionally occurred—carpopedal spasm, tingling and numbness of the hands and feet, and muscular cramps, etc. This was caused by the patients hyperventilating in an attempt to relieve a sensation of choking occurring during the attacks.

What was of greater significance, however, was the incidence of indifferent functional results following gastrectomy in some patients directly due to psychological causes. Few patients were free from difficulties in readjustment and occasionally these were allowed to grow until they occupied a dominant position in the mind, postponing complete return to health and detracting from the full value of the operation. The great majority resumed work within a few months with a renewed confidence, free from symptoms, and grateful to the surgeon for terminating many years of misery; a few, however, were not so fortunate and sought advise repeatedly on minor dietetic points, ability to undertake heavy work, etc., indicating clearly an uncertainty and anxiety for the future which need never have arisen.

An attempt was made to isolate the more important factors contributing to this state of mind, and these were found to be as follows :—

a. The disappointment often felt, but not readily expressed, at the recurrence of digestive and other symptoms following operation, and, because of them, the reasonable suspicion that operation had been a failure.

b. The patient's conviction that the stomach, even if diseased, was a vital organ and that its removal was of necessity attended by serious disabilities even if these were not symptomatically evident.

c. Uncertainty about the dietetic restrictions to be observed following operation, since opinions on this subject vary greatly.

d. The premature assumption of responsibilities to which they have become unaccustomed.

The importance of these factors and their prevention will be discussed later.

4. BILIOUS REGURGITATION AND VOMITING

These symptoms occur as components of the dumping syndrome, but were found occasionally unassociated with this syndrome. In the present series 28 patients suffered from bilious regurgitation and vomiting unrelated to the taking of meals. The complaint varied from one of mild bilious regurgitation to one of severe vomiting. The former occurred as an isolated phenomenon in 8 patients. Stooping and bringing up gas from the stomach appeared to be the chief precipitating causes. The symptom was regarded as of little importance to the comfort of the patient. In 19 patients, however, bilious vomiting occurred, which was classified according to causation as follows :—

a. In 10 patients it was considered to be psychoneurotic in origin. The characteristics and treatment of this type have already been dealt with.

b. In 7 patients it occurred only after the taking of a particular type of food, and was occasionally accompanied by diarrhœa. The foods blamed were cheese, meat, nuts, steamed fish (twice), and potatoes (twice). The vomiting came on within an hour of eating, was unaccompanied by any other symptom apart from diarrhœa and abdominal colic in three patients, and occurred each time the suspected food were eaten. A diagnosis of food allergy was made and further observations tended to confirm its accuracy. In two patients the suspected food was given in a disguised form and vomiting occurred one to two hours later. Other allergic symptoms occurred in 4 of the 7 patients, e.g., hay fever, urticaria, asthma, and angioneurotic œdema, and in one other a strong family history of asthma was admitted. It is possible that a tendency to food allergy is exaggerated by gastrectomy since one result of this operation is the passage of undiluted and chemically unaltered foods into the upper bowel.

c. In two patients the vomiting was regarded as obstructive in origin. This opinion was based on the clinical history of recurrent bouts of bilious vomiting of several weeks' duration, with complete remissions lasting for as long as six months. During the period of symptoms X-ray examination invariably revealed evidences of a partial high obstruction—delayed emptying of the gastric remnant and reverse peristalsis, culminating in some instances in vomiting of the barium. Gastroscopy was constantly negative in each patient. McNealy (1942) showed that kinking of the afferent and efferent bowel loops commonly followed subtotal gastrectomy and indicated methods by which this could be prevented. It is believed that these two patients suffered from intermittent kinking of the efferent loop of jejunum. Surgical treatment was considered, but postponed in the hope that a permanent remission would occur.

5. HYPOGLYCÆMIA FOLLOWING SUBTOTAL GASTRECTOMY

This was one of the less common syndromes encountered in the present series. It is believed that it has received undue emphasis in the literature of the subject, probably because the symptoms are

highly dramatic and were until recent times entirely unexpected.

Six patients were found who suffered from hypoglycæmic symptoms usually of a mild type. The clinical history had to be relied upon to a marked extent in coming to this diagnosis as attacks were infrequent and in two patients only was the diagnosis confirmed by blood-sugar estimations. Each patient showed a typical lag-curve response to the ingestion of 50 g. of glucose (*Table I, Cases* 7, 12, 20, 27, 30, 34), and the intervals following gastrectomy before hypoglycæmia developed were 3, 11, 6, 2, 17, and 3 months respectively. No patient experienced the syndrome within ninety minutes of a meal, the average interval being two hours. Hard physical exercise was the important precipitating factor in every patient and each had quickly realized that a small carbohydrate meal abolished the symptoms completely. The following two case records illustrate these points :—

Case 6.—J. B., aged 40, storekeeper.

Gastro-enterostomy for duodenal ulcer in 1924. Partial gastrectomy for stomal ulceration in 1943. Following the latter operation he experienced a severe dumping syndrome for several years which has now completely disappeared. Within a year following gastrectomy he experienced weakness, sweating, hunger, and palpitation before meals, particularly on physical exertion. These symptoms were rapidly abolished by sugar in any form, and he carried biscuits with him to work for this purpose. He has never lost consciousness but has occasionally had a strong feeling of apprehension during attacks and on one occasion he suffered from diplopia.

Case 7.—G. M., aged 47, cable jointer.

Subtotal gastrectomy for chronic gastric ulcer, Jan. 1, 1947. For the following three months he experienced a typical dumping syndrome after meals, which then disappeared. He then developed the following syndrome : Weakness, sweating, palpitation, and abnormal hunger appeared ninety minutes after meals, relieved by sugar in any form. If no sugar is taken they persist until a meal is eaten and then rapidly disappear.

A high-fat, low carbohydrate diet or ephedrine between meals would probably have eliminated the symptoms completely, but the attacks were considered to be too mild and infrequent to warrant such measures and were treated with complete success by explaining their nature to the patients and encouraging them to carry a few lumps of sugar about with them to use in emergencies.

6. DEFICIENCY SYNDROMES FOLLOWING GASTRECTOMY

It is well known that anæmia occasionally follows subtotal gastrectomy. This is usually of iron-deficiency type, but may rarely be due to deficiency of the intrinsic hæmopoietic factor. Seventeen patients in the present series developed moderate iron-deficiency anæmia following operation and all responded to large doses of iron. It is worthy of note that fifteen of these patients were women and that koilonychia was present in none. No patient developed megaloblastic anæmia.

Two other deficiency syndromes were met with. Two male patients exhibited mild scorbutic symptoms rapidly responding to ascorbic acid—follicular hyperkeratosis, bleeding gums, and tender subcutaneous hæmatomata. Questioning of these men revealed the fact that their diet previous to operation had been seriously deficient in vitamin C. The remaining deficiency syndrome was more common and warrants greater consideration. It was found that many patients following gastrectomy suffered from painful legs and not a few of these showed œdema of the ankles. Plasma-protein estimations in these patients were invariably normal and closer clinical investigation occasionally revealed the presence of areas of hyperæsthesia in the legs, associated with diminished and unequal tendon-jerks. A diagnosis of peripheral neuritis due to deficiency of vitamin B_1 was made and confirmed in 9 patients by the rapidity of cure following its administration. The remaining 11 who were unaffected by vitamin B_1 improved slowly on symptomatic treatment—massage and exercises, supporting elastoblast bandages when œdema was present, etc. The latter group were regarded as cases of transitory idiopathic postural œdema and the pain they experienced was put down to muscular strain following a prolonged period of muscular disuse, as occurs after many illnesses.

DISCUSSION

Abnormal carbohydrate metabolism was present in approximately one-third of a series of gastrectomized patients. This was revealed by performing a standard glucose-tolerance test on each, and consisted of an abnormally rapid rate of absorption of carbohydrate due to precipitate emptying of the gastric remnant. Following the sudden elevation of blood-sugar consequent to this a much more important fall to hypoglycæmic levels occurred, severe enough to produce hypoglycæmic symptoms in approximately 75 per cent of patients showing the abnormality. This second or hypoglycæmic phase has been attributed to the development of increased insulin sensitivity in gastrectomized patients, analogous to that which occurs in normal and diabetic subjects when carbohydrate intake is maintained at a high level. Of 12 gastrectomized patients exhibiting hypoglycæmic symptoms during the performance of a standard glucose-tolerance test, however, only 1 showed evidence of increased sensitivity to insulin. Furthermore, a hypoglycæmic phase was well marked in glucose-tolerance curves from 2 patients out of a total of 6 shortly after gastrectomy, suggesting that insulin sensitivity, acknowledged to take some months to develop, was not a significant factor. From a consideration of these facts the conclusion was reached that an increased insulin response by the pancreas to the early postprandial hyperglycæmia more adequately explained the occurrence of hypoglycæmic symptoms in the great majority of gastrectomized patients, though increased insulin sensitivity could not be eliminated as a contributory factor in a few instances.

The dumping syndrome occurred in 75 per cent of the patients in the present series. In approximately 10 per cent it was severe enough to prevent the return to full employment for an average period of five months following operation ; in 30 per cent it was moderate in character but was, notwithstanding, a major factor in disturbing the mental equilibrium

so essential to a good functional result; and in the remaining 60 per cent it was transitory, mild, and of little significance, occurring only after unusually heavy meals. The only other significant factor in a consideration of its incidence was its milder character and shorter duration in those patients who had undergone subtotal gastrectomy for gastric ulcer or early gastric carcinoma. It appeared to have its greatest incidence and severity in patients who had undergone operation for duodenal ulcer.

In the great majority of cases it made its appearance within a few weeks of operation when the patients started to take more generous meals. In 10 per cent of cases it was delayed for several months, but the patients comprising this group were apprehensive in character and did not attempt to eat normal-sized meals until this period had elapsed. Its onset was never delayed longer than thirty minutes following a meal and its duration varied from twenty minutes to two and a half hours, with an average of one hour. Paradoxically, the more severe its character the shorter was its duration in some cases, as vomiting occurred earlier, giving prompt relief. The chief manifestations of the syndrome were a sensation of repletion, constant nausea culminating in bilious vomiting in 50 per cent of cases, extreme lethargy and drowsiness, and vasomotor disturbances. Less constant were diarrhœa, flatulence, and dizziness. In the differential diagnosis of the syndrome three conditions are specially important—stomal ulceration, nervous aerophagy, and neurotic vomiting. Stomal ulceration was suspected in many patients exhibiting a marked dumping syndrome, and further investigations, including X-ray examination of the stomach, gastroscopy, and fæcal occult-blood tests, were frequently deemed necessary. The differential diagnosis is not difficult if it is remembered that epigastric pain is *not* a component of the dumping syndrome. Patients exhibiting the syndrome, even in its severest form, never referred to the sensation of epigastric fullness as pain, and there are usually no better judges of what does, and what does not, constitute gastric pain. Alkalis relieve the pain of stomal ulceration but have no effect on the epigastric fullness of the dumping syndrome, and the character of a meal is more important than its bulk in producing symptoms in patients with the former condition. Neurotic vomiting is unassociated with other component symptoms of the dumping syndrome with the exception of nausea and sweating; occurs at irregular times after even small meals in patients showing other psychoneurotic tendencies; and is useful in avoiding added responsibilities. Nervous aerophagy produces every clinical feature of the dumping syndrome, and should be suspected as the major contributing factor when the syndrome occurs after small meals, when it is relieved only by the bringing up of large quantities of gas from the stomach, and when other psychoneurotic traits are present. The process of air swallowing can be watched prior to the onset of symptoms and cure can be obtained by explanation and suggestion. The diagnosis of what is virtually a clinical type of dumping syndrome produced by aerophagy is extremely important as it is the only type which can be completely and rapidly cured.

In considering the cause of the syndrome the following facts are important:—

1. The incidence of the dumping syndrome in the present series (75 per cent) is much higher than the incidence of lag-curve responses to standard glucose-tolerance tests. Furthermore, 46 patients exhibiting the syndrome had normal glucose-tolerance curves and therefore could not be said to have an abnormal tendency to hypoglycæmic attacks. It may be argued that the tests did not give an accurate picture of the incidence of this tendency since they were performed at rest, and it is well known that physical exercise in itself lowers the blood-sugar level and would therefore increase any latent tendency to hypoglycæmia. The validity of this argument cannot be denied, but if more natural conditions are postulated as a factor favouring an increase in the tendency to hypoglycæmia, then an incumbent factor must be admitted as favouring a decrease, viz., the nature of the meals eaten, which, in ordinary circumstances, are not composed of pure readily absorbed carbohydrate, but because of their mixed constituent foods and insoluble nature are followed by more stable blood-sugar levels.

2. The incidence and severity of the dumping syndrome is lower in patients undergoing gastrectomy for gastric ulcer or early gastric malignancy. It has been suggested that this is due to the fact that a larger gastric remnant is often left in these patients.

3. A study of the symptoms following the ingestion of meals varying only in bulk and carbohydrate content revealed the fact that bulk was the factor, above all others, which determined their severity. The carbohydrate content of the meal was of no importance in this respect. These observations were borne out by questioning the patients, who were well aware that the size of the meal was the important precipitating factor.

4. Blood-sugar estimations made at short intervals during typical dumping syndromes in 11 patients did not reveal hypoglycæmia, and intravenous glucose had no therapeutic value in 14 others.

5. The artificial distension of the upper jejunum with balloons reproduced the syndrome in 2 out of 4 patients. The more natural distension produced by air swallowing in 5 patients had an identical effect.

It is therefore suggested that the dumping syndrome is purely mechanical in origin, being produced by distension of the upper jejunum as a result of precipitate emptying of the gastric remnant. Hypoglycæmia is believed to play no part in the production of symptoms, and measures taken for their relief should not be based on this assumption. It is also believed that mechanical distension produces the local manifestations of the syndrome—epigastric fullness, nausea, and bilious vomiting—and that the general features—flushing, sweating, lethargy, and drowsiness—are reflex disturbances consequent to irritation of the upper jejunum, of which mechanical distension is only one cause. This belief is based on the fact that very hot or very cold meals, no matter how small, are capable of inducing the general features alone, probably by means of thermal irritation. An allergic reaction in the upper jejunum may also explain the rare occurrence of mild dumping syndromes in which general

manifestations predominate following the ingestion of small quantities of a specific article of food.

The substitution of six small meals in the day for three large ones adequately controlled the symptoms in many cases, but in some a period of rest following heavier meals was necessary, until, as usually happened, symptoms retrogressed spontaneously. Ephedrine, atropine, and olive oil were found to be of little value in treatment. Some patients, however, exhibited an intractable variety of the syndrome and did not respond to such simple measures. The question of aetiology was reconsidered in these patients, particularly the part played by distension of the afferent bowel loop, since entero-anastomosis between the afferent and efferent loops affected a cure in 3 patients, and at operation kinking or undue length of the proximal segment was discovered. It is in fact possible that this mechanism accounts for a considerable proportion of cases of the dumping syndrome, even of milder type, and further investigation of this hypothesis is being undertaken. Secretions which pour into the afferent loop from the pancreas and liver are augmented by a considerable secretion from the duodenal mucosa and it has been estimated that a total of 1–1½ litres may be produced in twenty-four hours (McNealy, 1942). It is obvious that an obstruction to the free drainage of these secretions would produce a considerable rise in pressure within the duodenal segment. Wangensteen (1941) has shown that the intraluminal pressure in the obstructed duodenum of a dog varied between 20 and 45 cm. of water. Recently Mimpriss and Birt (1948) attributed poor functional results following gastrectomy to three mechanical factors :—

1. A narrow stoma, occurring only after a Billroth 1 gastrectomy for duodenal ulcer.

2. Proximal loop filling occurring maximally in operations involving antecolic anastomosis of the proximal loop to the greater curvature of the stomach. This procedure creates a long proximal segment into which food readily passes. Symptoms very similar to those of the dumping syndrome are attributed to the duodenal distension so caused (Lake, 1948). It is recommended that this type of operation should be abandoned for this reason.

3. Delayed emptying of the proximal loop secondary to kinking or some other obstruction along its length. The symptoms produced are similar to those of the dumping syndrome with the addition of fatty diarrhœa as a result of the damming back of bile and pancreatic juices from the food passing through the small bowel.

It is clear from all this evidence that proximal-loop obstruction must be considered as a cause of persistent postcibal distress following gastrectomy. It is not difficult to understand how this may produce symptoms similar to those of the dumping syndrome. Postprandial bilious distension of the kinked loop would account for the sensation of epigastric fullness and reflex general effects such as drowsiness, sweating, and lassitude. If intraluminal pressure rose to such an extent that the obstructive agent was overcome, of if the obstruction was situated distal to the gastric stoma, a considerable quantity of bilious secretion would rapidly accumulate in the gastric remnant, inducing vomiting with immediate relief of symptoms. It is realized that these views are entirely unsupported by experimental evidence, but it is maintained that they afford a logical explanation of the clinical features, operative findings, and cure by entero-anastomosis in some patients.

Since this may well be the cause of a higher proportion of dumping syndromes than is at present thought, it becomes important to the surgeon to develop an operative technique which minimizes the tendency of the proximal bowel loop to become kinked or otherwise obstructed. Common sites and methods of production of obstruction are reviewed by McNealy (1942).

Afferent loop obstruction was regarded as one cause of the dumping syndrome for the simple reason that cure was achieved by entero-anastomosis in 3 patients. It should be admitted, however, that one other explanation of the cure affected by this procedure is possible which does not depend on the assumption that the syndrome is due to distension of the proximal segment. If mechanical factors are accepted as the basis of the dumping syndrome, particularly the distension of the efferent jejunum following precipitate emptying of the gastric remnant, then it should be realized that entero-anastomosis between the loops would also tend to prevent undue distension of the distal segment by providing a further channel of several hundred cubic centimetres capacity into which food may readily pass. In other words entero-anastomosis provides a reciprocal method of preventing distension of either loop and might be used routinely with advantage in performing subtotal gastrectomy in an attempt to minimize the incidence and severity of the dumping syndrome, were it not for the fact that the secretions of the proximal segment have a valuable prophylactic effect on the development of stomal ulceration and should not be diverted from the site at which they do most good. The operation should be reserved for patients with severe intractable dumping syndromes responding to no other form of treatment.

To summarize, it is believed that the dumping syndrome is due to sudden distension of the upper jejunum by food in the majority of cases ; that air swallowing is a neglected but potent aggravating cause of this distension in a few neurotic patients ; that some severe examples are due to intermittent or partial obstruction of the afferent bowel loop ; and that, finally, some mild cases in which the general features of the syndrome predominate may be due in part to thermal or allergic irritation of the jejunum.

Treatment is based on this mechanical hypothesis, and in mild and moderate cases the prescription of smaller meals followed by a short period of rest is uniformly successful. In severe cases responding to no other form of treatment and showing no tendency to recover spontaneously, entero-anastomosis between the efferent and afferent bowel loops may be considered.

Neurotic symptoms following gastrectomy were frequently encountered. Three syndromes were specially common—neurotic vomiting, nervous aerophagy, and brief syncopal attacks. The first two were often misdiagnosed as dumping syndromes and the third was regarded as hypoglycæmic in origin until further investigations were undertaken. It was also pointed out that psychological maladjustment

was a potent cause of prolonged convalescence or incomplete return to health in some patients. Apprehension because of the reappearance of digestive symptoms following operation, the conviction that serious disability attended the removal of the stomach, dietetic difficulties, and the anxieties of prematurely accepted responsibilities, were the chief sources of worry encountered in the present series. Adequate treatment for the majority of digestive upsets following gastrectomy is available, and each patient should be told clearly that temporary digestive symptoms following operation are common, usually disappear spontaneously within a year, and do not mean that recurrent peptic ulceration is imminent. They should also be convinced that absence of the stomach is compatible with perfect health, and it is often of value in this respect to enlist the aid of a patient who has shown no symptoms following operation, and has returned to full vigour and working capacity. It is believed that too little attention has been paid to the diet of recently gastrectomized patients. The confidence of the patient is undermined by constantly varying dietetic advice. It is suggested that for six months following gastrectomy meals should be small and frequent, of high calorie value, neither very cold nor very hot, and of limited fluid content. This advice, given as a routine, would minimize the discouraging symptoms which larger meals often produce at this time and allow a quicker return of confidence. Finally, gastrectomized patients should not readily accept heavy responsibilities until the difficult early phase following operation has passed. This applies mainly to patients who previously suffered from peptic ulcer, the majority of whom are anxious to resume their previous activities at an early date. They are in fact over-anxious to do so and it is remarkable what little effort is required to rehabilitate them. Nevertheless, this is often accomplished too quickly and the readiness with which previous responsibilities are assumed often masks a very real fear that their accomplishment will prove physically impossible. This will only be admitted on intimate questioning and is apt to be overlooked. It is suggested, therefore, that more gradual rehabilitation, particularly in patients with psychoneurotic traits, should be practised. It may be argued that functional upsets are only encouraged by such mental placebos and that patients should be encouraged to face realities. It is believed, however, that the anxiety of a patient with peptic ulcer differs from that of an anxiety state. In the former it acts as a driving force frequently translated into action from which, it is contended, the patient requires temporary protection, while in the latter it acts as a brake to activity and encourages introspective idleness. It is probable that nothing will change the anxious, ambitious, obsessional mental pattern of the patient with peptic ulcer, but this does not mean to say that he should be allowed to become his own executioner. Simple psychological guidance is necessary and greatly benefits those who have undergone partial gastrectomy. There is surely no more interesting field for psychiatric work than one in which cure of a psychosomatic illness is attempted by surgical means.

Failure to gain weight following gastrectomy was common and was regarded anxiously by most patients. It occurred maximally in those who suffered from severe symptoms and was due in part to inadequate feeding. This was corrected by substituting concentrated foods of high calorie value for bulky foods of low calorie value. It must be admitted, however, that increase in weight was slow and averaged no more than a few pounds each month. Seven patients were encountered in whom failure to gain weight was unaccompanied by digestive symptoms. Five of them exhibited a mild degree of steatorrhœa, which was regarded as an important causative factor. It is suggested that steatorrhœa is not uncommon in gastrectomized patients and may be a potent cause of failure to gain weight. Further investigation of this important point is obviously required.

Bilious vomiting, apart from its occurrence as part of the dumping syndrome, was encountered in approximately 5 per cent of patients in the present series. It was commonly psychoneurotic in origin, but in some patients the possibility of an allergic basis was considered as it only occurred after one particular type of food. Clinical and radiological evidence of obstruction of the efferent bowel loop was obtained in 2 patients, and it is likely that each will require surgical treatment in the future.

Hypoglycæmic attacks occurred in 6 patients and were all of mild type. Their time of onset dated from three to seventeen months after operation and symptoms appeared approximately two hours after meals. In the present series they were a very minor cause of disability following gastrectomy, and it is believed that their importance has been greatly exaggerated. No patient required to alter his mode of life because of them, although it is admitted that none were of the severity recorded by Barnes (1947), Gilbert and Dunlop (1947), and others. It is stressed that neurotic syncopal attacks are often misdiagnosed as hypoglycæmia and occur with equal frequency.

Deficiency syndromes also were not a major cause of disability. Iron-deficiency anæmia occurred principally in women, and rapidly responded to treatment. One syndrome—neuritic pains in the legs and feet, muscular cramps, ankle œdema, and sensory disturbances—responded to injections of vitamin B_1. No patient showed evidence of intrinsic hæmopoietic factor deficiency.

In conclusion, lest this report should appear unduly pessimistic, it should be pointed out that its subject is the patient who has a poor functional result following gastrectomy. In the great majority the operation was entirely successful in its object of removing a potent source of misery and a danger to life. Some patients paid for this more than others in the form of various digestive upsets, usually transient, but all were agreed, once the nature of the symptoms had been explained and confidence had been regained, that health had been immeasurably improved, and, indeed, the only source of regret in many was that gastrectomy had not been performed years previously.

SUMMARY

1. An analysis of the symptoms following subtotal gastrectomy in 124 patients is undertaken.

2. The symptoms were found to fall into six

groups in the following order of incidence and importance :—

a. The dumping syndrome.
b. Failure to gain weight after operation.
c. Psychoneurotic symptoms.
d. Bilious regurgitation and vomiting.
e. Hypoglycæmic attacks.
f. Deficiency syndromes.

3. The incidence, aetiology, clinical features, and treatment of the dumping syndrome is discussed. It is suggested that its origin is mechanical and that it is sometimes based on obstruction of the afferent bowel loop.

4. Failure to gain weight after operation is attributed to inadequate feeding in the majority of patients secondary to the presence of digestive symptoms. Steatorrhœa was found in five underweight patients and is regarded as one cause of the syndrome.

5. The psychological difficulties of the patient who has undergone recent gastrectomy are discussed and methods of dealing with them suggested.

6. Hypoglycæmia was found to be a relatively unimportant cause of postcibal distress in the present series.

7. The incidence and aetiology of abnormal carbohydrate metabolism in gastrectomized patients as judged by the results of a standard glucose-tolerance test are investigated and discussed.

This work was carried out mainly during the tenure of a Nuffield Research Grant. My thanks are due to Professor C. F. Illingworth and Mr. W. J. M. Brandon for allowing me to investigate their patients, and to Professor J. W. McNee for his help and encouragement in the preparation of this paper.

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MODERN TRENDS IN HARE-LIP AND CLEFT PALATE SURGERY*

WITH A REVIEW OF 500 CASES

BY MICHAEL C. OLDFIELD
SURGEON, THE GENERAL INFIRMARY AT LEEDS

" *He gave Man speech, and speech created thought,*
Which is the measure of the universe; "
SHELLEY, P. B., *Prometheus Unbound*, Act ii, sc. 4, l. 72.

A CHILD who is without intelligible speech rarely acquires a lively, bright intelligence. More often, his character is warped by complexes, born from an overwhelming consciousness of his own inferiority. When a child with lip and palate cleft is allowed to go to school before his speech and looks have been restored, life there can be made almost unbearable by the constant mimicry of his comrades.

* A Hunterian lecture delivered at the Royal College of Surgeons of England on Feb. 10, 1949.

AETIOLOGY

Incidence.—One baby in about 600 born comes into this world with lip and palate cleft, and so in England there are probably about sixty thousand people who were born with these clefts. At present we do not know how to prevent this because we know so little about the cause.

Causation..—After years of investigation only two factors of significance have emerged: (1) Inheritance; (2) Maternal malnutrition or disease during the first three months of the pregnancy.

1. *Inheritance.*—A family history of the deformity was revealed in only 11 per cent of our 500 cases. The mother's side of the family was affected in 6 per cent, the father's in 3 per cent, and both were affected in 0·4 per cent; the defect occurred in brothers and sisters in 1·6 per cent. These figures have, however, little scientific importance owing to the world-wide reticence of a mother-in-law in respect of her forebears' shortcomings. This is scientifically deplorable, but in other respects laudable.

Fogh-Andersen (1942 and 1946) made a careful study of the families of 703 patients in Denmark during the occupation and found that 30–40 per cent of these patients had near relations with similar deformities. From this investigation he concluded that inheritance was the most important aetiological factor. The laws of human heredity are so complicated that, if we accept this, as I think we should, we are still far from the solution of the problems of the causation and prevention of hare-lip and cleft palate. It seems probable that a recessive gene is responsible in most cases, but not all individuals who inherit the requisite genetic constitution suffer from the deformity, and the classical Mendelian figures are not found. It may be that additional influences have to exert their effect before the deformity becomes manifest. Exactly what other factors are involved is not known. Some may be genetic also, and sex genes or relative growth-rate genes may be involved. Some may be purely environmental, such as malnutrition or diseases, like rubella, occurring early in the mothers' pregnancy. It is probable that the double dose of recessive factor makes the developing embryo unduly sensitive at critical periods, especially during the first three months of gestation, to what would otherwise be unimportant variations in uterine environment. From the figures of incidence of the deformity in affected families, apparently only one-quarter to one-half of those bearing the double dose of recessive, develop the deformity.

Sex seems to be an additional genetic factor of considerable importance, for when considering all types of the deformity, boys are much more often affected than girls (*Table I*). There is evidence

Table I.—Sex Distribution in All Types of Cleft Lip and Palate

	Male		Female	
	No. of Cases	*Per-centage*	*No. of Cases*	*Per-centage*
Fogh-Andersen (1000 cases)	606	60·6	394	39·4
Present series (500 cases)	302	60·4	198	39·6

Ratio: Male : Female
3 : 2

also that at least one of the gene pairs causing an extensive cleft may be partially sex linked (Haldane, 1936; Mather and Philip, 1940).

Hare-lip with or without cleft palate is more common in boys, and a positive family history was found in 27 per cent of Fogh-Andersen's hare-lip cases and 40 per cent of his hare-lip and cleft-palate cases. When inherited, the cleft-bearing factor appeared to be a recessive. *Isolated cleft palate, on the other hand, occurs more commonly in girls* and was a hereditary character in only 19 per cent of his cases, when it was a dominant but with varying degrees of penetration (*Table II*).

Table II.—Sex Distribution amongst Cases of Hare-lip alone, Cleft Palate with Hare-lip, and Isolated Cleft Palate

	Fogh-Andersen (625 cases)		Present Series (500 cases)	
	Male	*Female*	*Male*	*Female*
Hare-lip	90	48	53	35
Hare-lip and cleft palate	257	103	168	77
Cleft palate	43	84	81	86

Hereditary prognosis: Fogh-Andersen's investigation upon the hereditary factor led him to certain conclusions which might help other surgeons to answer some of the difficult questions so often put to them by parents. For instance:—

a. If two apparently normal parents have a child with hare-lip or hare-lip and cleft palate, and there has been no previous history of these deformities on either side of the family, the chance of deformity in their next child is slight, probably about 5 per cent.

b. If one parent has a hare-lip or hare-lip and cleft palate the chance of deformity in the children is only about 2 per cent, but if a deformed child has already been born to them, the chance is raised to 15 per cent for subsequent children.

c. Only on the rare occasions when both parents have hare-lip or hare-lip and cleft palate or both have isolated cleft palates is the incidence amongst the offspring high. The only possible contra-indication to marriage or having a family is when these conditions prevail.

2. *Maternal Malnutrition and Disease.*—Animal experiments suggest that maternal malnutrition or disease during the first three months of pregnancy may 'uncover' a pair of recessive genes bearing the cleft-causing factor. Breeders discovered many years ago that when the diet of lionesses or bulldog bitches during the early weeks of pregnancy was deficient in fresh meat, a high percentage of the cubs or puppies were born with clefts in lip or palate. Adequate supplies of fresh meat reduced the incidence to negligible proportions. (Vevers, 1940.)

In man little experimental data is available, but it was accepted that in previous generations, before rationing, hare-lip and cleft palate occurred most frequently in children of poor people and was rare in the families of the well-to-do. Recently, however, Fogh-Andersen found no evidence of this in Denmark.

a. Rh sensitivity: Rh sensitivity does not appear to play any important part in the causation of hare-lip and cleft palate (Stephenson, 1948), and it may be significant that 40 per cent of our cases were

first-born while 60 per cent were the result of subsequent deliveries.

b. Incidence of the different types of cleft: The lip seems to be cleft about as commonly as the palate, for in Veau's, as well as Fogh-Andersen's series, each composed of 1000 cases, roughly a quarter of the patients had hare-lip alone, a quarter cleft palate alone, and a half hare-lip and cleft palate together (*Table III*).

Table III.—INCIDENCE OF CLEFTS IN THE LIP AND PALATE

	Veau (1000 cases)	Fogh-Andersen (1000 cases)		Present Series (500 cases)	
	Approx. Per cent		Per cent		Per cent
Hare-lip alone	25	275	27·5	88	17·6
Hare-lip and cleft palate	50	514	51·4	245	49·0
Cleft palate alone	25	211	21·1	167	33·4

I. HARE-LIP

Anatomical Types of Hare-lip.—Hare-lips may be classified as unilateral or bilateral, either of which may be complete or incomplete. The degree of deformity of nostril, lip, and prolabium varies considerably within each of these groups and many ill-defined subgroups can be recognized, but it is interesting to note that in all reported series unilateral hare-lip is much more common on the left than on the right, and no explanation has been found for this (*Fig.* 232).

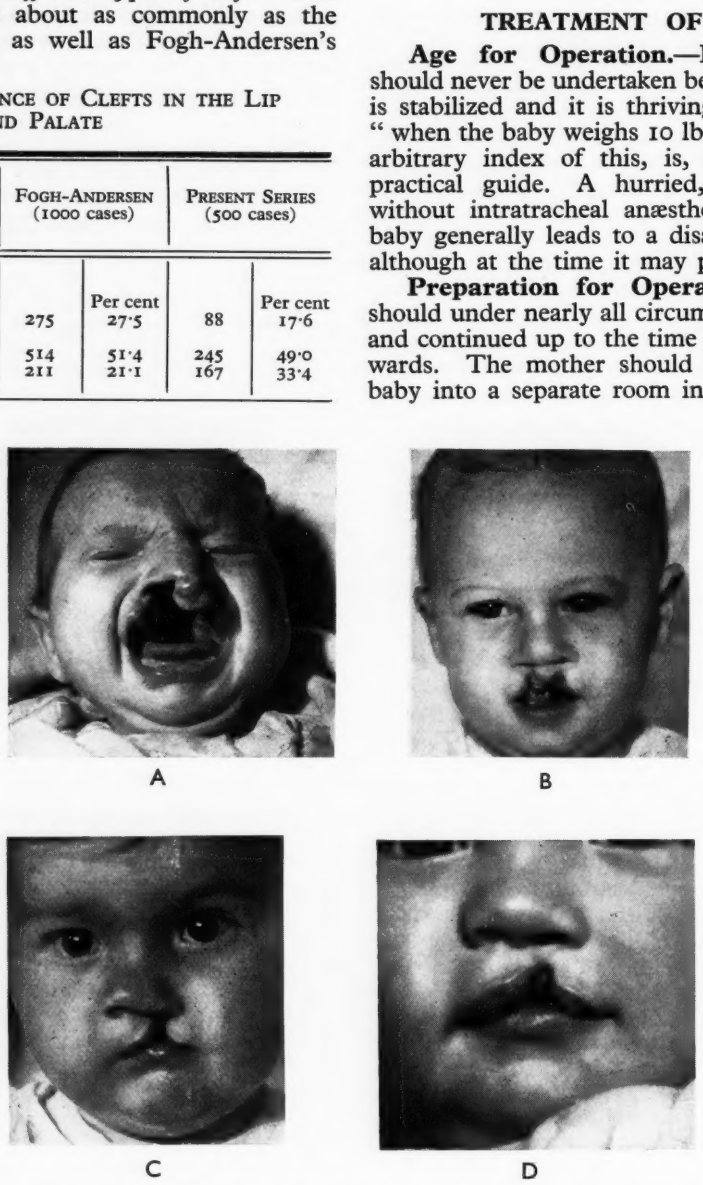

FIG. 232.—Photographs showing: A, Complete bilateral hare-lip; B, Incomplete bilateral hare-lip; C, Complete unilateral hare-lip; D, Incomplete unilateral hare-lip.

TREATMENT OF HARE-LIP

Age for Operation.—Repair of a hare-lip should never be undertaken before the baby's feeding is stabilized and it is thriving and gaining weight: "when the baby weighs 10 lb.", a traditional though arbitrary index of this, is, nevertheless, a useful practical guide. A hurried, imprecise operation, without intratracheal anæsthesia, upon a newborn baby generally leads to a disappointing final result, although at the time it may please the parents.

Preparation for Operation.—Breast feeding should under nearly all circumstances be encouraged and continued up to the time of operation and afterwards. The mother should be admitted with her baby into a separate room in the hospital, whether the baby is 'on the breast' or not, because however the baby is fed, the mother is usually the best person to look after it. If this is impossible to arrange, because the mother cannot leave her home or other children, the baby should still be admitted to a small room away from direct or indirect contact with other babies and should *never be admitted to a general infant's ward*. For, if a hare-lip baby is treated in the ward, there is always a danger of gastro-enteritis in the post-operative period and tragedies will occur. These infections may be extremely virulent, and as we are so profoundly ignorant of their nature, no specific antidote is available. It seems, however, to be generally agreed

that it is an infection which is usually contracted in a hospital ward and is carried directly or indirectly from another infant. Breast-feeding ensures a valuable immunity, and the danger of cross-infection can be avoided if the baby is isolated in a separate room without any contact with other babies or with nurses who look after them.

When arrangements can be made for the mother to be admitted with her baby, forty-eight hours should be allowed for them to settle down and accustom themselves to their new surroundings. During this time, the baby is observed to see that he is taking his feeds satisfactorily and that he has no loose green stools. Everyone who enters the room wears a mask and it is wise to make certain that neither the mother nor the baby is starting a 'cold'. An ordinary feed is given two hours before operation, and plaster cuffs are put over the elbows.

Operation

Anæsthesia.—Intratracheal anæsthesia is given through a small plastic tube, using oxygen and ether on an open circuit by the Philip Ayre (1937) method, with a throat pack in position.

1. For Unilateral Hare-lip and Nostril Deformity.—With the exception of the very simple type where there is merely notching of the lip and red margin, unilateral hare-lip is associated with nostril deformity in both complete and incomplete types (*Fig.* 233). In the repair, therefore, of these deformities it is of vital importance to remedy this

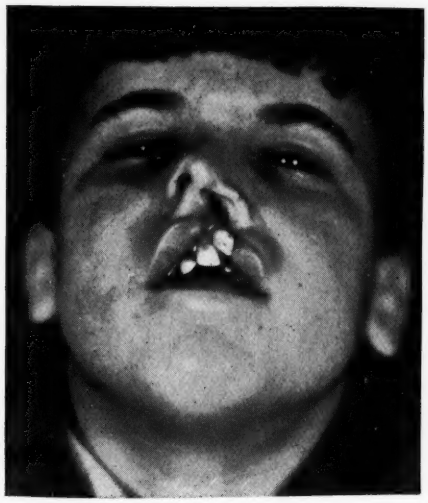

FIG. 233.—Photograph of a patient showing nostril deformity associated with unilateral hare-lip.

nostril defect before closing the lip. The main elements of nostril distortion requiring adjustment are :—

a. Widening and depression of the nostril base.

b. Depression of the dome of the nostril, especially in the medial third.

c. Loss of external convexity of the alar rim, which in severe cases may be stretched almost horizontally over the lip cleft.

d. Deviation of the columella base to the opposite side.

In general the degree of nostril deformity varies with the width of the lip cleft and the nostril is not so much deformed in partial clefts as it is in the wide complete ones.

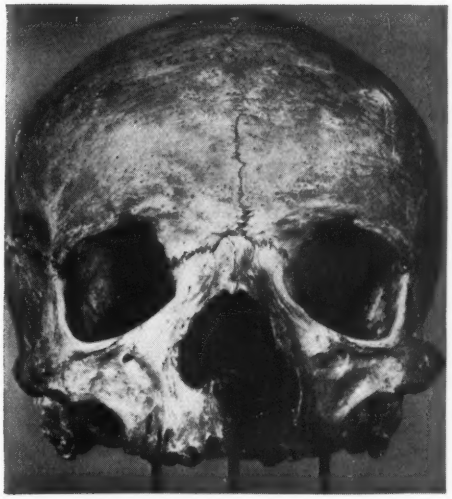

FIG. 234.—Skull of patient with unilateral hare-lip to show bone deformity of nostril.

The underlying defect in the bone appears to be the cause of the soft-tissue deformity of the nostril. (*Fig.* 234.)

The soft tissues are not usually defective, but merely distorted, and so they require to be freed from the bone and then re-shaped, before being sutured into correct position. This principle of filleting the soft tissues from the defective skull bones as the first stage in the operation, is used not only in repair of the nostril and lip, but also, later, when the palate is repaired.

a. Nostril repair : Wide mobilization is carried out through an ample incision in each upper buccal sulcus. On the affected side the whole of the upper lip and the cheek, as far as the infra-orbital margin, are mobilized. The affected nostril is separated completely from the bony margin of the pyriform opening ; whilst the base of the nostril on the uncleft side is also freed from the bone. The base of the columella is then separated from the displaced anterior nasal spine. By extending the incision in the upper buccal sulcus into the nostril on the cleft side and by inserting a pair of curved scissors, the lining of the nostril is freed from its attachment to skin and cartilage as far as the tip of the nose. If mobilization is complete, the nostril can then be repaired so that it is similar to that on the normal side. (*Fig.* 235.)

b. Lip repair : The repair of the lip is a comparatively simple procedure in comparison with that of the nostril. It can be done by straight excision of the margins of the defect according to the traditional Rose principle, or a small flap composed of skin and red margin can be preserved on the cleft side and used for repair of the lower border, on the Mirault principle.

I prefer straight excision, using Denis Browne's (1935, a) chisel-knife for the minor incomplete

clefts; but for the major complete clefts I have found that the lip often tends to be rather long and tight near the mucocutaneous line, unless the small flap is saved on the outer side and used for repair of the lower border. A short lip with a full roll at the lower border gives a more pleasing result, and can be attained, I think, more consistently if a small flap and a zig-zag suture line is used. Blair and Brown (1930) have described many useful practical details of technique when applying the Mirault principle for lip and nostril repair, but their original operation was unnecessarily complicated and the simplified technique of Brown and McDowell (1945) is more practical.

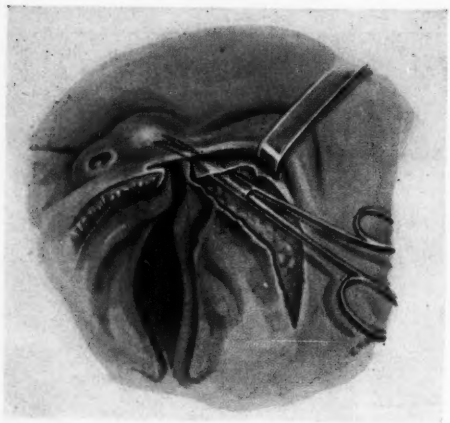

FIG. 235.—Repair of unilateral hare-lip, showing mobilization incision and scissors freeing the lining of the affected nostril.

Technique of Hare-lip Operation.—Before starting the operation, 'guide marks' are tattooed on the skin with Bonney's blue (*Fig.* 236).

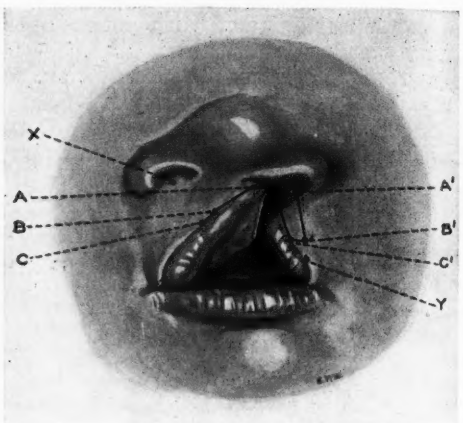

FIG. 236.—Mirault-Brown operation for repair of unilateral hare-lip, showing the 'guide marks' on the skin before starting the operation: 1, A is tattooed with a Schimmel's point dipped in Bonney's blue and lies within the skin just above the mucocutaneous line on the level with the base of the columella on the medial aspect of the cleft. 2, X is marked with a pen in the normal nostril in the same relationship to the columella base as mark A on the cleft side. 3, A^1 is tattooed on the cleft side in a similar relationship to the alar base as X bears to the ala on the normal side. When A is sutured to A^1 the base of the two nostrils should then be symmetrical. 4, C is tattooed just within the skin above the mucocutaneous line on the medial side of the cleft at the point where the vermilion is still of full thickness, but just before it begins to thin out. This point usually lies medial to the ridge of the philtrum on the uncleft side of the lip. 5, Y is marked with a pen on the mucocutaneous line on the latersl side of the cleft, using measuring callipers, so that it is the same distance from A^1 as C is from A. 6, C^1 is tattooed on the mucocutaneous border of the lateral side of the cleft at the point where the vermilion on this side is still of full thickness, but just before it starts to thin out. 7, B^1 is on the line A^1–Y equidistant from Y and C^1, and it is usually about one-third or one-quarter of the distance from Y to A^1. The distance between C^1 and B^1 is measured with callipers. 8, B is tattooed just within the skin above the mucocutaneous line on the medial side of the cleft so that it is the same distance from C that B^1 is from C^1.

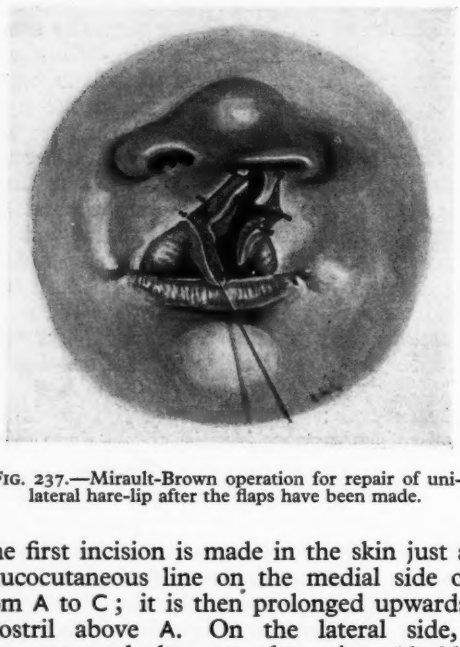

FIG. 237.—Mirault-Brown operation for repair of unilateral hare-lip after the flaps have been made.

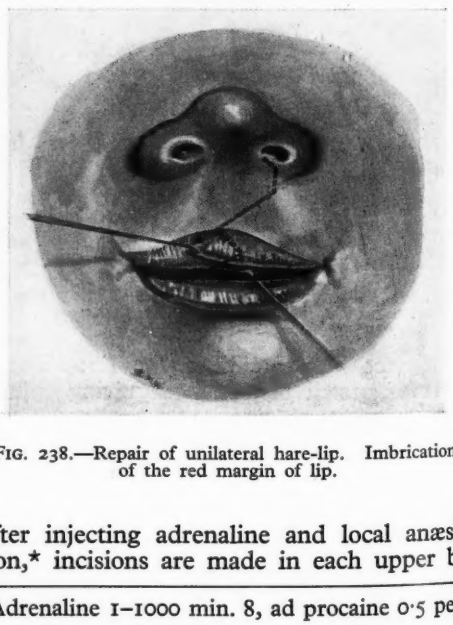

FIG. 238.—Repair of unilateral hare-lip. Imbrication of the red margin of lip.

The first incision is made in the skin just above the mucocutaneous line on the medial side of the lip from A to C; it is then prolonged upwards into the nostril above A. On the lateral side, skin incisions are made between the points A^1, B^1, and C^1; from C^1 the incision is carried upwards along the mucocutaneous line into the lining of the nostril on the cleft side.

After injecting adrenaline and local anæsthetic solution,* incisions are made in each upper buccal

* Adrenaline 1–1000 min. 8, ad procaine 0·5 per cent oz. 1.

sulcus to free the lip, nostrils, columella, and the cheek on the affected side and separate them from their attachments to bone. These incisions are packed with rolled gauze soaked in 1–1000 adrenaline. The skin incisions, already outlined on the front of the lip, are then deepened by transfixion with a No. 11 Bard Parker knife which divides the whole thickness of the lip. On the medial side there is

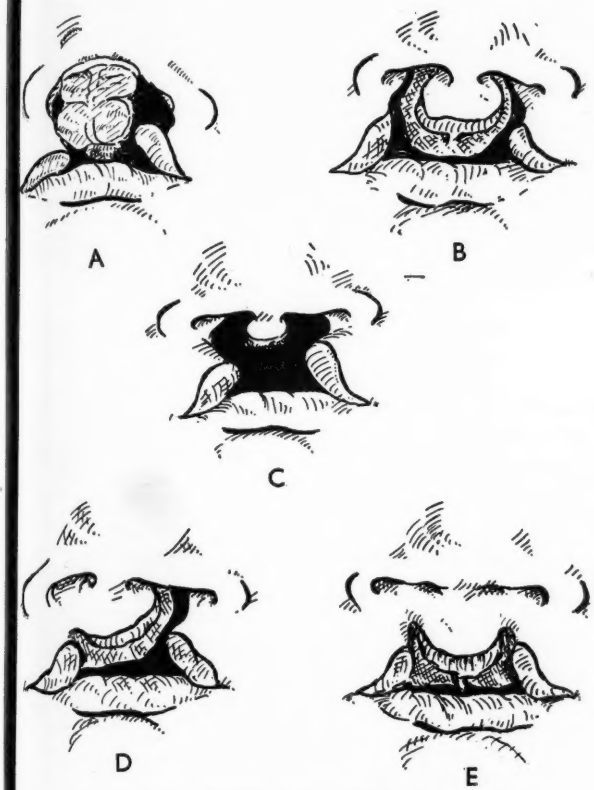

FIG. 239.—Five common types of bilateral hare-lip.

now a red mucosal flap, and on the lateral side a skin and mucosal flap; both of these are drawn downwards and the coronary arteries near the angle of each are ligatured with very fine silk (*Fig.* 237) The deep tissues beneath the alar base and columella are approximated with a fine silk suture. The buccal mucosa of the under surface of the lip is closed with mattress sutures of chromic catgut. The muscle of the lip is sutured with fine silk so that the points A and A^1 and B and B^1 and C and C^1 are approximated. The skin is sutured with gossamer silkworm gut, threaded on small eyeless needles.

When repairing the vermilion border, a vertical suture line should be avoided, otherwise notching or a redundant papilla may result. Imbrication prevents this, and a V-flap of red mucosa on the medial side is made to fit into a V excision on the lateral side. With the lateral vermilion flap held in front of the medial one, the necessary incisions are made with fine-pointed scissors (*Fig.* 238).

When the operation is finished, an adjustable Denis Browne traction bow is applied over the lip and attached to each cheek with adhesive strapping.

2. For Bilateral Hare-lip.—In the most severe cases (*Fig.* 239, A) of bilateral hare-lip, where there is gross anterior tilting of the premaxilla, a two-stage programme of repair is usually a wise precaution. At the first operation the premaxilla is replaced in correct alinement with the maxilla and the soft tissues on one side of the cleft are repaired. Six weeks later the other side is closed.

Re-alinement of Premaxilla.—At the first operation, to effect this re-alinement of the premaxilla, a longitudinal incision is made along the undersurface of the front of the septum. Lateral mucosal flaps are raised on each side, and a rectangular block of bone is removed with nibbling forceps. The medial margins of the alveoli of the maxilla and the lateral margins of the premaxilla are pared. The premaxilla is then drawn back into correct position and fixed there by a stainless-steel wire suture. This is inserted with a sharp bone awl across the front of the premaxilla and through the alveolar process of each maxilla and tied on the buccal aspect.

Soft-tissue Repair.—The soft tissues of the lip and nostril are repaired by direct excision or by making a small lateral vermilion border flap. If the bilateral hare-lip is not associated with gross premaxillary tilting and the cleft is complete on one side and incomplete on the other, direct excision on the incomplete side of the cleft, and a flap repair on the other side, give a satisfactory result (*Fig.* 240).

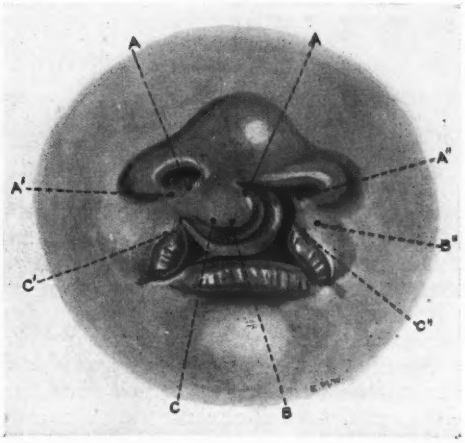

FIG. 240.—'Guide marks' used for repair of a bilateral hare-lip. On the right side the cleft is incomplete, and the direct excision technique is used; on the left, where the cleft is complete, a lateral mucocutaneous flap is used.

In all cases of double hare-lip, the lateral elements of the lip are approximated, as in normal development, below the prolabium. The red border of the prolabium is turned backwards and used for lining the posterior surface of the lip: it takes no part in the reformation of the visible red margin.

Elongation of the Columella.—After primary repair of a complete bilateral hare-lip the columella tends to be short and the tip of the nose is drawn down (*Fig.* 241). This deformity requires adjustment when the patient is three or four years old. The tip of the nose is freed and the prolabial skin is advanced into the columella by a V–Y lengthening procedure (Brown and McDowel, 1947). (*Fig.* 242.)

RESULTS

Deaths.—Amongst 224 patients treated by operation for repair of hare-lip deformities 4 have died within a month of the operation—a mortality of 1·8 per cent. Two of these patients died at home. They appeared to be quite fit on discharge from hospital, but soon after arriving home gastro-enteritis developed suddenly and was due to an infection which had probably been contracted from another baby in the ward. Following these deaths, separate small rooms have been provided in hospital and no deaths have occurred since.

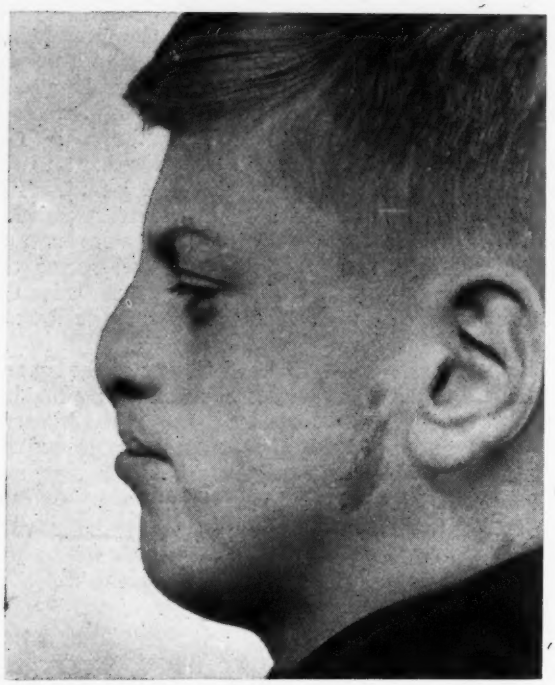

FIG. 241.—Photograph showing depression of the tip of the nose after repair of a complete bilateral hare-lip during infancy.

Anatomical Results.—No attempt has been made to classify the anatomical results, as no accurate scientific criterion of this seemed possible. In forming an estimate of the result the opinions of the parents and their friends are involved as well as the surgeon. In general it may be said that the parents are usually delighted and, as a group, they are amongst the most grateful of all patients' friends, whereas the surgeon is rarely satisfied. He sees some evidence of the hare-lip deformity afterwards and compares his results with the normal child. Fortunately the mother compares the result of his work with the condition of the baby before operation. But this should be no solace to the surgeon, because he should remember that the neighbours and 'friends' of the family also estimate the result by comparison with a normal baby.

In hare-lip surgery some progress has been made in recent years, but we have still far to go, much farther, I think, than in the surgery of cleft palate.

II. CLEFT PALATE

Anatomical Types of Cleft Palate.—Veau's (1931) classification of cleft palate into four types has the great practical merit of simplicity and has now been adopted in most countries (*Fig.* 243):—

Type I: Cleft of the soft palate only.
Type II: Cleft involving part of the hard palate as well as the soft palate.
Type III: Unilateral cleft of the alveolus as well as the hard and soft palate, usually associated with unilateral hare-lip.
Type IV: Bilateral cleft of the alveolus as well as the hard and soft palate, usually associated with bilateral hare-lip.

Table IV.—INCIDENCE OF THE FOUR TYPES OF CLEFT PALATE

	VEAU (1000 cases)	PRESENT SERIES (412 cases)	
	Percentage	*Cases*	*Percentage*
Type I	20·8	57	13·8
Type II	30·8	133	32·2
Type III	38·8	162	39·5
Type IV	9·6	60	14·5

In Type II there often seems to be a defect in the soft-tissue elements associated with a congenital shortening of the palate. There is a wide gap in the front of the cleft and sometimes there is also an absence of the palatal bones (*Fig.* 244), whereas in Types III and IV there is usually no lack of soft tissue, but merely a failure of fusion in the midline. In a fairly high proportion of Type II cases the mandible as well as the maxilla seems to be imperfectly developed, and the access at operation in

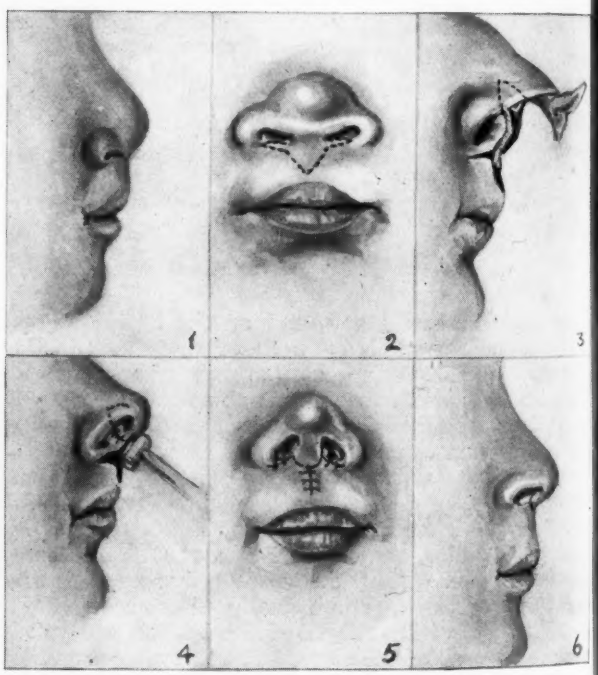

FIG. 242.—Operation for elongation of the columella and elevation of the depressed tip of the nose after repair of a complete bilateral hare-lip in infancy.

these cases is sometimes limited by the resulting mandibular regression (*Fig.* 245).

Amongst 7 of these cases of mandibular regression, there were 2 newborn babies in whom the tongue periodically fell back, behind and above the lateral elements of the soft palate, and sudden attacks of asphyxia occurred when the baby turned on to its back (*Fig.* 246). These babies require very careful supervision and feeding during the first three months. They should always be kept lying on the side by a sandbag placed along the back. An œsophageal feeding-tube may occasionally be necessary in the early weeks of life. Gradually, as the baby learns to suck and the strength of the tongue and jaw muscles improve, the so-called ' tongue-swallowing ' becomes progressively less frequent and ceases after the third month.

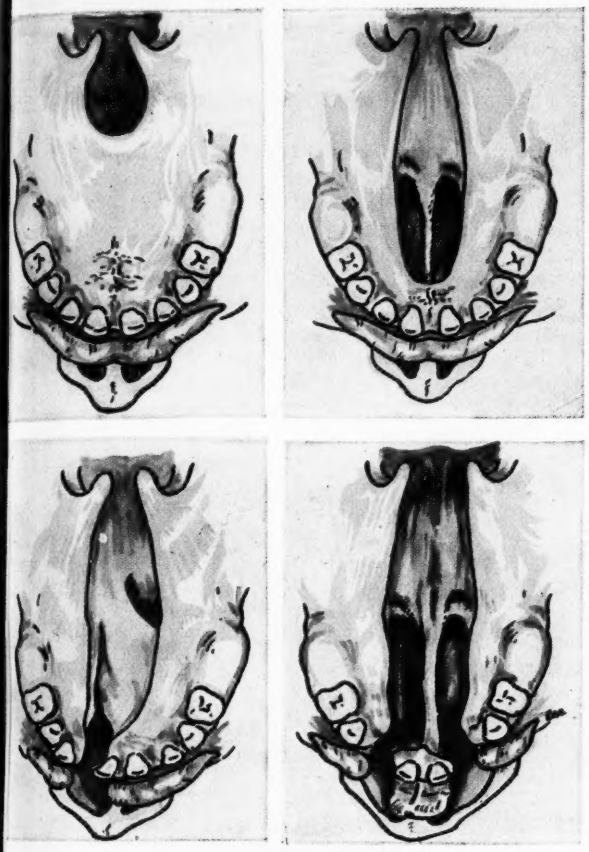

FIG. 243.—Veau types of cleft palate.

FIG. 245.—Patient with mandibular regression associated with cleft palate, Veau Type II.

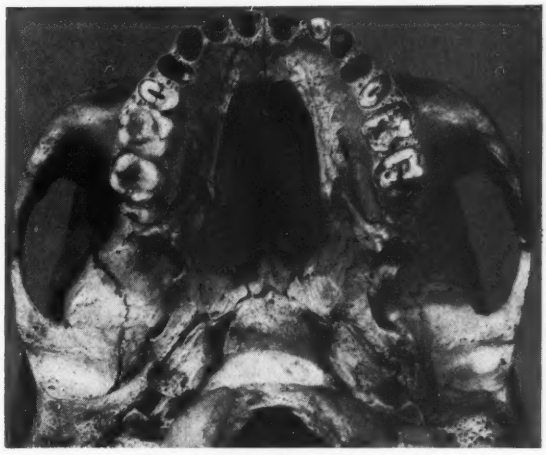

FIG. 244.—Skull showing Veau Type II cleft palate with absence of palatine bones.

Submucous Clefts.—In a proportion of cases the bone and muscles are cleft more extensively than the mucosa or skin. Veau (1931, 1938) described these conditions of the lip and palate, but he did not mention an occult type of cleft palate, described recently by Mengele (1939) which we have encountered on two occasions. In these two patients speech was typically ' cleft palate ' in type but there was no cleft in the buccal or nasal mucosa. The soft palate in the midline was thin and membranous and contained no muscle; the levator sling was incomplete and the nasopharyngeal sphincter incompetent. It seems probable that this type of cleft palate is often not recognized and the speech defect is considered to be functional instead of organic. It would seem that the correct treatment is first to excise the thin membrane, then to define the muscle layers by undercutting the mucosa: the soft palate is repaired by suturing the nasal mucosa, the muscles, and finally the buccal mucosa.

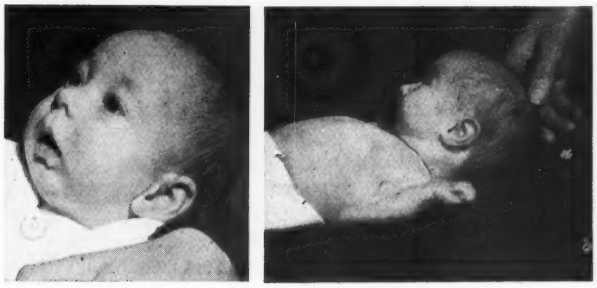

FIG. 246.—A baby who was a ' tongue swallower ' associated with Veau Type II cleft palate and mandibular regression.

The Mechanism for Closure of the Naso-Pharyngeal Isthmus.—Anatomists are unfortunate because their descriptions of muscles are usually based upon dissections they have made upon subjects partially putrefied and then pickled. Surgeons are in some ways more fortunate, because they can

observe the natural anatomy whilst they are at work amongst muscles which are alive and often only too active.

The nasopharyngeal isthmus is closed by a sphincter mechanism composed of a series of overlapping muscle slings (Denis Browne, 1935, b; Oldfield, 1941), arranged in three dimensions around the nasopharynx. During speech, the valve is opened and closed with lightning rapidity, occlusion being effected by elevation of the *middle third* of the soft palate into contact with Passavant's ridge and the lateral walls of the nasopharynx (*Fig.* 247).

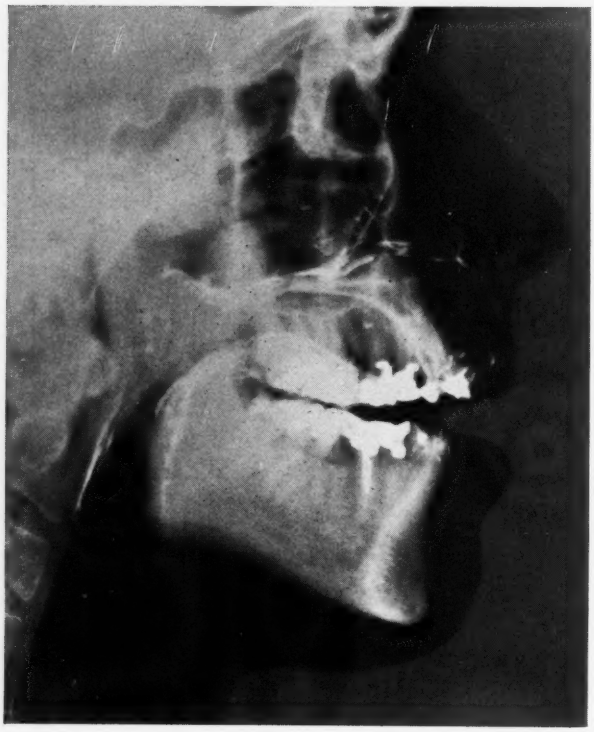

FIG. 247.—Radiograph (Dr. A. S. Johnstone) showing the elevation of the middle third of the soft palate when the nasopharyngeal valve is closed during the articulation of an oral consonant sound 'K'.

This arching of the soft palate is effected by the levator sling, which consists of two muscle bellies working from fixed points above, on the base of the skull, and connected below by the tendinous aponeurosis of the soft palate. When the muscle bellies contract, the middle third of the soft palate is raised into occlusion with the posterosuperior and lateral walls of the pharynx. The levator palati sling is therefore a *single* unit with two lateral motors. The usual anatomy book description of two levator palati muscles is sadly misleading.

The salpingo-pharyngeus and the muscles of the posterior pillars (the pharyngo-palati) complete the occlusion on the lateral aspect, by drawing the salpingo-pharyngeal folds and the posterior pillars upwards and inwards. The 'horse-shoe muscle-fibres' which, on contraction, raise Passavant's ridge on the posterosuperior wall of the pharynx, are inserted into the soft palate on each side and pass almost horizontally around the pharynx. These fibres were considered by Passavant (1862, 1863, 1869, 1878, 1879) and Whillis (1930) as belonging to the upper part of the superior constrictor, whereas Wood Jones (1940) has more recently shown that they represent a separate and distinct muscle, the 'palato-pharyngeus'.

It seems probable that the ridge of Passavant is kept erect constantly during speech by tonic contraction of these fibres, and acts, according to Wardill's description (1928), as a 'valve seating' for the upper surface of the middle third of the soft palate. Only when there is a lull in speech do these fibres relax, and anyone can confirm upon himself Wardill's observation that there is a " feeling of slow relaxation which normally occurs in the posterior pharyngeal wall on cessation of speech ".

The tensor palati acts normally as an antagonist to the levator sling and is used in swallowing to press food boli downwards and backwards. If, however, in a cleft palate patient the fulcrum at the hamulus is destroyed (Billroth, 1889), the tensor can be converted into a second levator sling which is a great help in speaking (*Fig.* 248).

During normal conversation, complete nasopharyngeal occlusion is required to raise the necessary pressure of air in the mouth for articulation of explosive and friction sounds. When the palate is cleft, the levator sling is incomplete in the midline, the nasopharyngeal valve is incompetent, and oral consonant sounds are difficult to pronounce.

One of the main objects for achievement, therefore, when repairing a cleft palate, should be to restore, and also to reinforce, the sling action of the levator. Thus an active and muscular soft palate, which can be arched rapidly into contact with the posterosuperior wall of the nasopharynx, is provided. The nasopharyngeal valve will then be effective and normal consonant sounds produced (*Figs.* 249, 250). It is more important to provide a rapidly arching and muscular soft palate than a long and flaccid one. *Length is not so important as muscularity and mobility.*

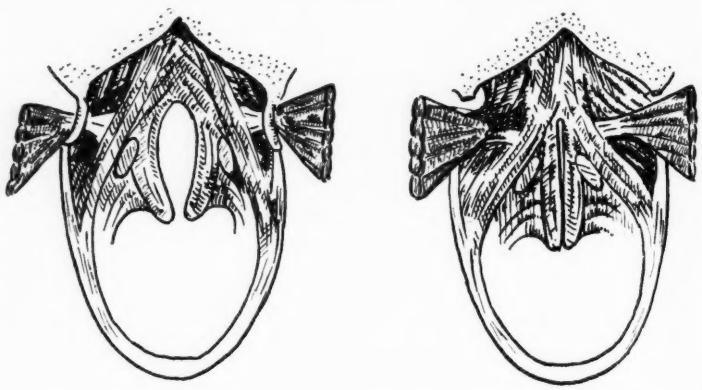

FIG. 248.—Diagram showing tensor palati from above, before and after the hamulus is fractured.

TREATMENT OF CLEFT PALATE

Age for Operation.—The palate should be repaired when the child is between one and two years old, usually at about eighteen months, so that the palate and nasopharyngeal valve are already effective when the patient learns to speak. Operations for repair of the palate in infants under a year old are not followed by any better speech results, but are associated with a mortality of over 10 per cent compared with a mortality of under 2 per cent for operations during the second year.

FIG. 249.—Radiograph (Dr. A. S. Johnstone) taken during the articulation of a nasal consonant sound ' M '—the naso-pharyngeal valve is open.

FIG. 250.—Radiograph (Dr. A. S. Johnstone) taken during the articulation of an oral consonant sound ' S '—the naso-pharyngeal valve is closed.

Contra-indications to Operation.—Operation for repair of the palate is advisable in all child patients unless there is evidence of gross mental deficiency or an uncompensated congenital heart lesion. Many patients with compensated heart lesions can be treated if they are carefully prepared for operation by an experienced cardiologist, and four of our patients who had compensated congenital heart lesions were treated successfully by operation. A few adults still come for treatment whose soft palates have been lost irretrievably by sloughing after a series of unsuccessful operations in childhood, and in these an obturator must be used. In the majority of ' grown-ups ', however, the whole palate can be repaired and, in many others, enough tissue remains for repair of the soft palate and only the hard palate needs replacement by an obturator.

Anæsthesia.—Anæsthesia is induced by ethyl chloride and continued by oxygen and ether, on open circuit, given through a plastic intratracheal tube inserted over the tongue, but without a throat pack. A special Harris type of metal connector* is used to prevent compression of the intratracheal tube between the lower incisor teeth and the tongue-piece of the gag.

Position, Lighting, and Suction.—A soft pillow is placed under the head and shoulders, so that moderate extension of the neck is obtained with adequate support for the head. The table is raised and the surgeon stands above the patient's head. The surgeon who sits has not the freedom of movement that is required in this operation. If a well-focused scialytic light is available, no head-light is required. A Kilner's ' Shadwell gag ', with wire spring ligature carrier, is inserted, and the mouth is cleaned with cetavlon.

So that a clear view can be assured throughout, two suction motors are essential. The first is connected by pressure tubing and a Y glass connector to two plastic catheters which are inserted through the nostrils into the nasopharynx above the soft palate. The second motor is connected by tubing to a metal suction end, which is handled by the assistant. Periodically, as in a brain operation, clots are washed away by saline irrigation with a small bulb syringe.

Technique of Operation.—In the technique of operation we have followed the principles first

* Made by Chas. F. Thackray Ltd., Park Street, Leeds.

laid down by Veau (1931) and extended later by Wardill (1937) and Kilner (1937). During the last two years certain modifications have been added which seem to have simplified the technique* and made the repair more substantial.

PRIMARY CASES

1. Paring the Edges of the Cleft in the Soft Palate and incising the Edges of the Hard Palate —On the right side, the central part of the medial edge of the soft palate is grasped with fine-toothed dissecting forceps held in the left hand. A pointed No. 11 Bard Parker knife with long handle (No. 7), held in the right hand, is used to transfix and then pare the inner edge of the soft palate and uvula. The inner edge of the hard palate is incised with a No. 15 blade along the line of junction of the red nasal mucosa and pink buccal mucosa. This incision is prolonged forward as far as the anterior extremity of the cleft. On the left side, the medial edge of the soft palate is grasped with the dissecting forceps held in the right hand and transfixed and pared with the knife held in the left. The medial edge of the hard palate on this side is incised in the same way as on the right (*Fig.* 251).

2. Lateral Relaxation Incisions and Fracture of Hamular Processes.—To reduce bleeding and assist subperiosteal elevation of the soft tissues, local anæsthetic solution with adrenaline* is injected over the bone of the hard palate, and along the line of the pterygo-mandibular raphe into the lateral pharyngeal space of Ernst. An incision is then made with a No. 15 knife along the line of the pterygo-mandibular raphe and continued forward along the inner side of the gums of the temporary molar and premolar teeth. Posteriorly this incision is deepened with a straight elevator, through the pterygo-mandibular raphe until the glistening silver tendon of the internal pterygoid is seen in the outer aspect of the wound. The hamulus, the position and size of which varies considerably, is identified by dissection with the elevator or by palpation with the little finger. The hamulus is then fractured through its base by pressing upon its outer side with the elevator (*Fig.* 252).

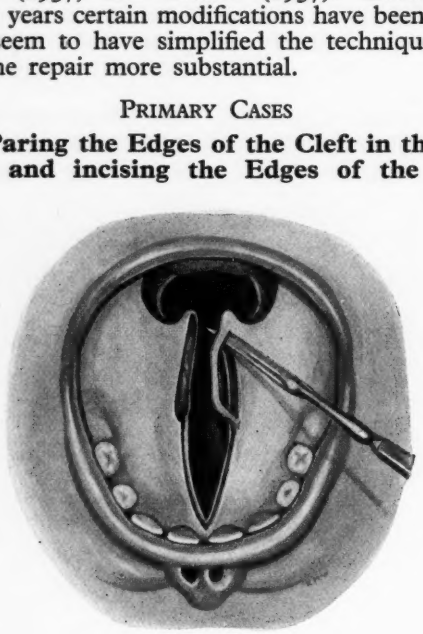

FIG. 251.—Paring the edges of the soft palate and incising the edges of the hard palate defect.

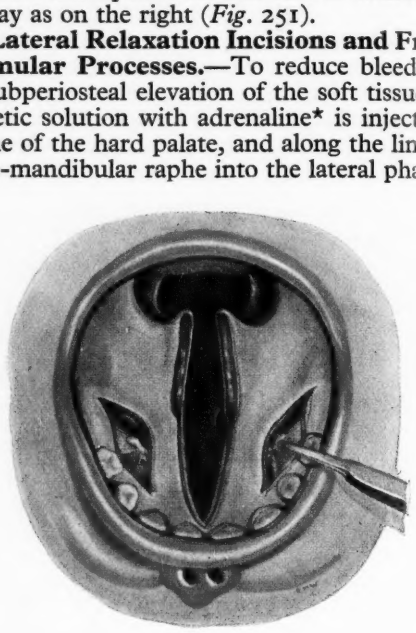

FIG. 252.—Lateral relaxation incisions and fracture of the hamular process.

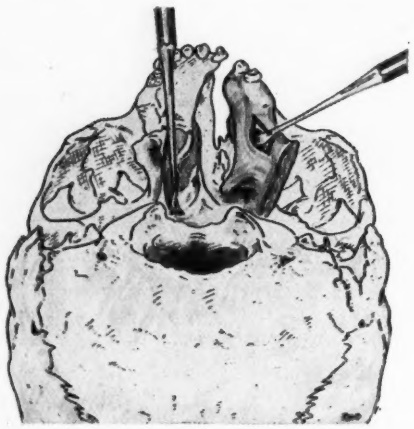

FIG. 253.—Diagram showing the elevator passed along the inner side of the internal pterygoid plate to mobilize the side wall of the nasopharynx.

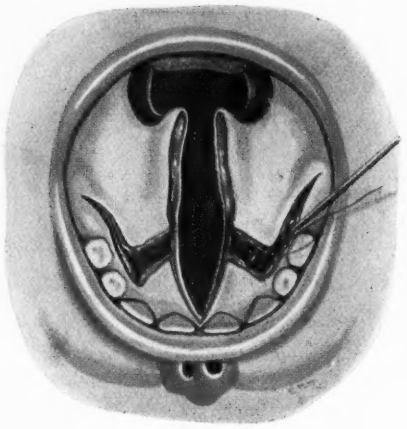

FIG. 254.—Anterior oblique incision, ligature of communicating branch between anterior and posterior palatine arteries.

* A cinematograph film was shown during the lecture to demonstrate the technique of the operation.

* Adrenaline 1–1000 min. 8, ad 0·5 per cent Procaine oz. 1.

It is important to effect a clean fracture through the base, so that the elevator can then pass subperiosteally into the next plane of cleavage, which extends along the inner side of the internal pterygoid plate, as far as the base of the skull and the roof of the pharynx (*Fig.* 253). The side wall of the naso-pharynx is thus freely mobilized, and rolled gauze, soaked in adrenaline solution, is packed temporarily into the lateral pharyngeal space.

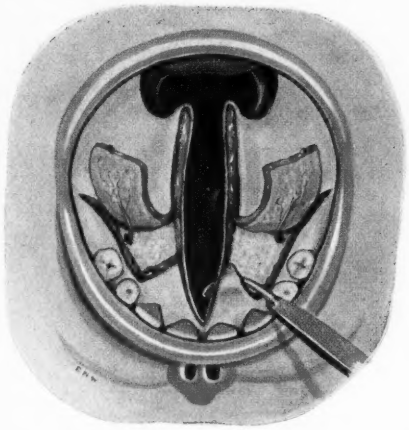

FIG. 255.—Elevation of short anterior buccal flaps. The long posterior buccal flaps have been elevated.

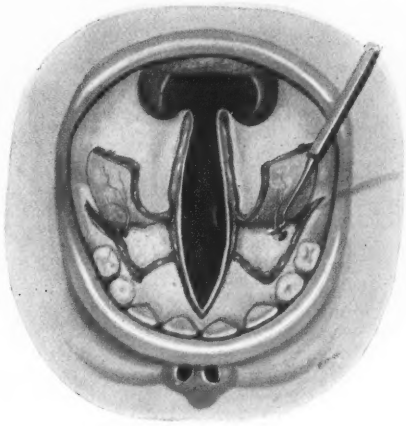

FIG. 256.—Mobilization of posterior palatine artery.

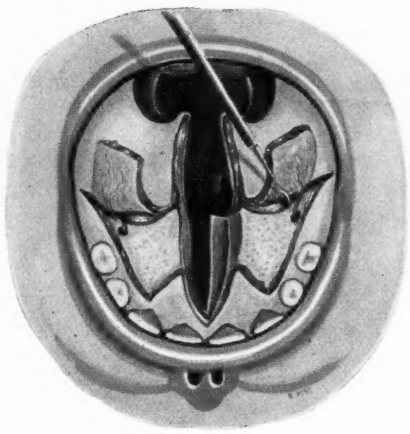

FIG. 257.—Mobilization of lateral nasal flaps in a unilatera cleft. The elevator has been inserted from the medial aspect of the posterior palatine artery into the lateral plane of cleavage made by the dissection along the inner side of the internal pterygoid plate.

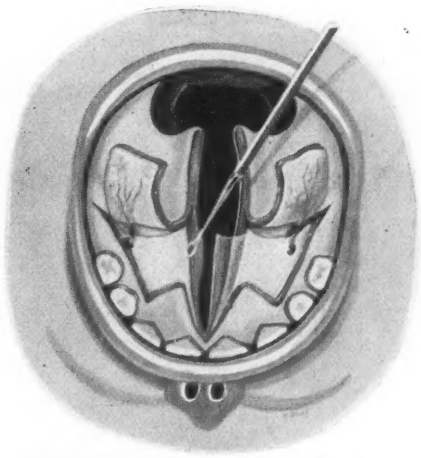

FIG. 258.—Mobilization of lateral nasal flaps in a unilateral cleft. The elevator has been passed above the horizontal plates of the palate bone and maxilla and is being drawn forward between the bone and nasal mucosa. As it passes forward it frees the nasal mucosa from its attachment to bone along the line of the cleft in the hard palate. The septal flap has already been raised on the opposite side, i.e., on the patient's right side.

In front, the relaxation incision is deepened with a straight elevator along the lateral aspect of the hard palate. Near the maxillary tuberosity, firm fibrous bands attaching the buccal mucosa to the posterior part of the alveolus require division with a few strokes of the right-angled knife. A right-angled elevator is inserted into the anterior part of the incision and is used to raise the hard-palate flap from the bone. It emerges at the medial edge of the cleft in the hard palate through the incision already made during the first stage of the operation.

3. Anterior Oblique Incisions and Ligature of the Communicating Branch between Anterior and Posterior Palatine Arteries.—An oblique incision is now made across the hard palate, starting at the edge of the cleft about ¼ in. in front of the junction of the hard and soft palate, and passing outwards and forwards to the anterior extremity of the lateral relaxation incision. The oblique incision extends to bone in the inner two-thirds, but only through the mucosa in the outer third. The antero-lateral angle of the posterior flap is freed and held backwards, so that the large communicating branch between anterior and posterior palatine arteries can be isolated for some distance by blunt dissection with a small curved elevator. Two ligatures are then passed around the artery with a fine fully-curved aneurysm needle, and tied as far apart as possible, so that the vessel can be divided, with pointed scissors, between the ligatures (*Fig.* 254). Failure to secure this vessel before division may be followed by considerable hæmorrhage during the next stage of the operation, and bleeding may persist after the patient returns to the ward.

4. Elevation of Short Anterior Buccal Flaps.—The short anterior triangular buccal flaps are then lifted off the bone with a curved elevator (*Fig.* 255).

5. Elevation of the Long Posterior Buccal Flaps and Mobilization of the Posterior Palatine Artery.—The anterior margin of the posterior flap is drawn backwards while a straight elevator is used, in a bloodless subperiosteal plane, to lift the flap from the palatal process of the maxilla and the horizontal plate of the palate bone. The posterior edge of the bony palate is carefully defined, and the attachment of the palatal aponeurosis is separated from it. A firm fibrous band from the

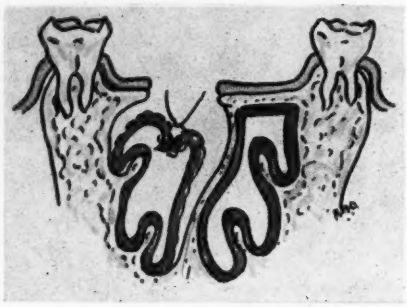

FIG. 259.—Transverse section through hard palate showing lateral nasal and septal flaps in a unilateral cleft.

azygos uvulæ muscle is divided at its attachment to the posterior nasal spine with a small hooked knife. Near the lateral side of the flap the posterior palatine artery is seen as it emerges from the posterior palatine canal. The artery is mobilized on its posterior aspect by dissection along the posterior margin of the horizontal plate of the palate bone, using a small curved elevator, passed through the lateral relaxation incision (*Fig.* 256): mobilization is then completed with the elevator inserted

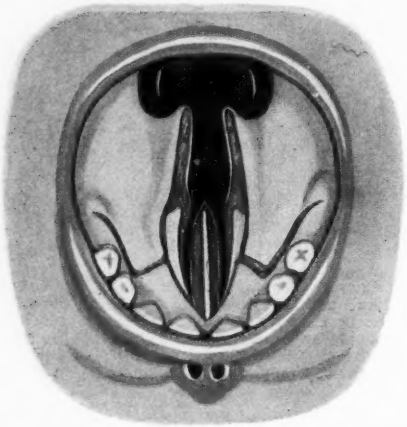

FIG. 260.—Septal and lateral nasal flaps in a bilateral cleft.

from the medial aspect. The lateral plane of dissection along the inner side of the internal pterygoid plate now communicates with the medial plane of dissection along the posterior edge of the bony palate. A small spoon-shaped* elevator is inserted from the medial aspect along this plane of cleavage medial to the internal pterygoid plate, and the mobilization of the nasal mucosa from the side

* ' Spoon ' refers here to a golf spoon not a teaspoon.

wall of the nasopharynx and floor of the nose is started from this point.

It is never necessary to divide the posterior palatine arteries when repairing a cleft palate, for, if the arteries are correctly mobilized from the canal, they are not a cause of lateral tension on the posterior flaps, and, moreover, they provide the main blood-supply to them. Sloughing of large areas of the posterior flaps may follow division of these vessels, but if they are left intact, sloughing rarely occurs.

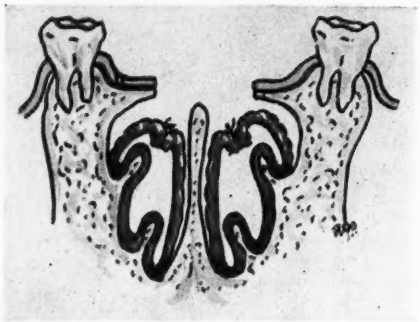

FIG. 261.—Transverse section through hard palate showing lateral nasal and septal flaps in a bilateral cleft.

6. Mobilization of Lateral Nasal Flaps.—During the next stage of the operation, the mucosa lining the floor and side of the nose is freed from bone and mobilized so that it can be drawn inwards without tension and sutured to a flap raised from the nasal septum.

A curved spoon-shaped elevator is inserted from the medial side along the posterior margin of the bony palate, passing behind the mobilized posterior palatine artery into the lateral pharyngeal space,

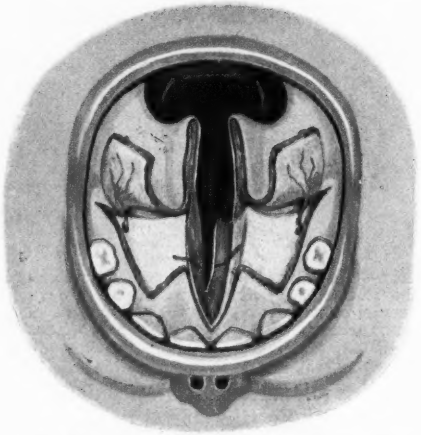

FIG. 262.—Suturing nasal mucosa of hard palate in unilateral cleft.

already opened up by the dissection through the lateral relaxation incision, along the inner side of the internal pterygoid plate (*Fig.* 257). The elevation is continued forward from this point and the nasal mucosa is separated from the posterior border of the bony palate and the posterior nasal spine. The elevator passes above the horizontal plates of the palatine bone and maxilla and is drawn forward between the bone and the nasal mucosa; as it passes forward, it frees the nasal mucosa from its attachment

to bone along the line of the cleft in the hard palate (*Fig.* 258). If the nasal mucosa is closely attached to this edge of bone, the plane of cleavage can be demonstrated previously if a small sharp periosteal elevator is used to press the periosteum and nasal mucosa off the inner edge of the bone.

In double clefts, when the septum is not fused with either maxillary process, a similar lateral nasal flap is raised on the other side.

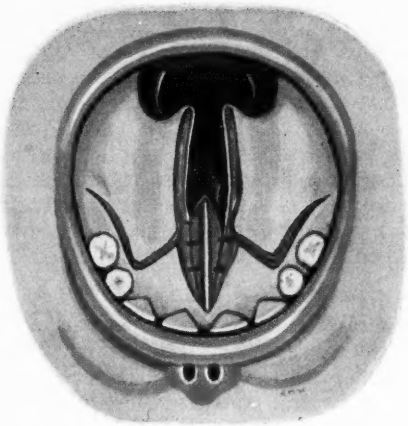

FIG. 263.—Suturing nasal mucosa of hard palate in bilateral cleft.

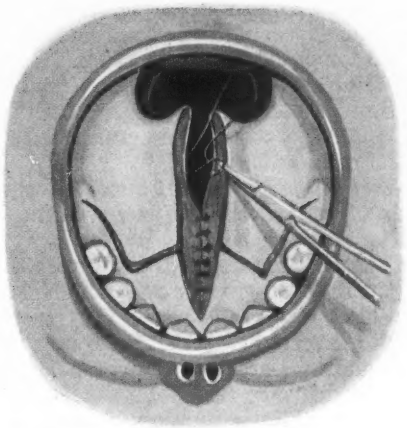

FIG. 264.—Suturing nasal mucosa of soft palate.

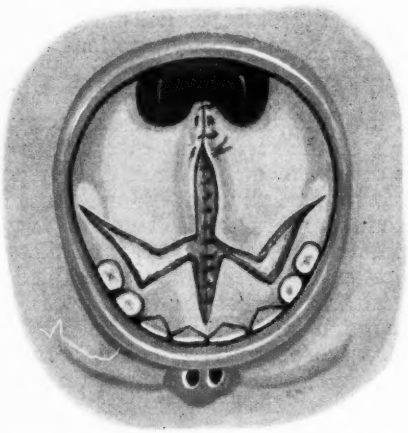

FIG. 265.—Suturing of muscles of soft palate and buccal mucosa with mattress sutures.

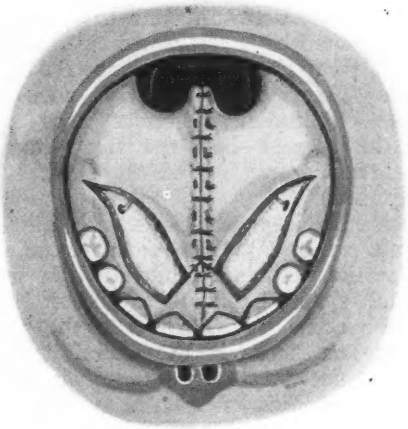

FIG. 266.—Sutured buccal mucosa.

7. Mobilization of Nasal Septum Flaps.—*a. Unilateral cleft*: In a unilateral cleft when the nasal septum has fused with the maxillary process on one side, the septum passes obliquely down and outwards to its maxillary attachment. A flap of nasal mucosa can be raised therefore under direct vision from the exposed surface of the septum, and, when freed, this flap can be drawn over to meet the lateral nasal flap (*Fig.* 259).

b. Bilateral cleft: In a bilateral cleft when the septum is not attached to either maxillary process, it descends vertically into the cleft; a longitudinal incision is made along its lower free margin and a flap of septal mucosa is raised from each side of it (*Figs.* 260, 261).

Suturing.—Before inserting any sutures it is wise to test the mobility of all the various flaps, by drawing them together in pairs to the midline, to see that they meet without tension. For most of the stitches I use 3/0 chromic catgut threaded on small, fully-curved cleft-palate needles* mounted on an Adson's needle-holder.

a. Nasal mucosa: The first stitch is inserted into the nasal mucosa near the anterior end of the cleft so that the knot can be tied on the inferior surface. In unilateral clefts the lateral nasal flap is stitched to the septal flap (*Fig.* 262), and in bilateral clefts each septal flap is sutured to each lateral nasal flap (*Fig.* 263). At the junction of the hard and soft palate the method of inserting the stitches is changed. The needle enters the nasal mucosa of the soft palate on its upper aspect and is brought out in the line of the cleft. The needle then re-enters the nasal mucosa of the opposite side from the buccal aspect and comes out on the upper surface, where the knot is tied (*Fig.* 264). By inserting these stitches in two separate movements, a little more time is spent, but a stronger

* Cleft-palate needle $\frac{11}{16}$ circle bend, length $\frac{3}{4}$ in., diameter $\frac{3}{8}$ in., with round body and short triangular point, made by Messrs. Chas. F. Thackray Ltd., Park Street, Leeds.

hold can be taken of the nasal mucosa above the soft palate. When the posterior end of the upper surface of the soft palate is reached, the two halves of the uvula are drawn forward and one or two mattress sutures are put in to ensure eversion of the nasal mucosa at this point, where inversion tends to occur.

b. Uvula: The uvula itself is repaired with fine eyeless sutures.

c. Muscles of the soft palate: It is most important to approximate the muscles of the soft palate either by a separate layer of buried sutures or by using an 'end on' type of mattress suture for the buccal layer of stitches. The first deep bite of these stitches passes through the muscle as well as buccal mucosa, while the second, superficial bite includes only buccal mucosa (*Fig.* 265).

d. Buccal mucosa: In the buccal closure, single stitches should, however, alternate with the 'end on' or lateral mattress sutures. In this way eversion is ensured without ischæmia of the suture line. The buccal mucosa over the hard palate is lengthened in the midline by the Wardill (1937) method of suturing the inverted 'V' incision by an inverted-Y line of stitches. The mucosa which lay originally along the medial two-thirds of the posterior edge of the anterior oblique incision, is rotated inwards and stitched in the midline to the corresponding mucosal edge of the opposite side.

The two short anterior triangular flaps of buccal mucosa are rotated inwards and their inner margins are approximated by lateral mattress sutures, because, here again, there is a great tendency for the margins to invert. The apex of the large triangle formed when the two small triangular flaps have been sutured, is inserted into the small inverted 'V' at the anterior extremity of the sutured posterior flaps (*Fig.* 266).

The mouth is washed out with saline from a small bulb syringe to remove all loose clots. In every case the packs are removed from each lateral pharyngeal space before the patient leaves the theatre; because I believe that the practice of retaining gauze packs in the lateral incisions after operation is not only quite unnecessary if a correct method of mobilization has been used, but is a dangerous and a dirty habit. Penicillin powder is blown into each lateral recess with an insufflator. A stitch is passed through the dorsum of the tongue and penicillin cream is applied to the corners of the mouth to prevent them from getting sore.

Treatment of Secondary Clefts

Ten years ago one patient out of three who came to us for treatment had had previous unsuccessful operations elsewhere. During the last year only about one patient in six has had a previous operation for repair of the palate. In the majority of these secondary cases there is enough tissue left to repair the whole of the palate if careful and often tedious mobilization is carried out. If, however, much soft tissue has been lost, then it is wise to use the remaining tissue to repair the soft palate by a Gillies-Fry (1921) procedure, and often in these cases a Wardill's (1923, 1933) pharyngoplasty is required, if there is likely to be incompetence of the nasopharyngeal valve without it. A pharyngoplasty is never advisable when repairing a palate as a primary procedure, because an actively contracting Passavant's ridge is preferable to the stiff, inert, fibrous bar which results from a pharyngoplasty.

POST-OPERATIVE TREATMENT

The patient goes to the ward lying on his side after the cough reflex has returned. Penicillin 500,000 units is given intramuscularly twice a day for a week after the operation. Watery fluids are given by mouth as soon as the patient is conscious. After twenty-four hours purées and minced foods are given and each feed is followed by a drink of cold water to act as a mouth-wash. Ice-cream is allowed, but ordinary milk is not given, as it tends to clot on the stitches. In young children no local treatment is advisable, but in older children and adults, penicillin lozenges are useful and sodium bicarbonate mouth-washes are given after each feed. The patient usually goes home between the tenth and fourteenth day after operation.

AFTER-TREATMENT

1. Speech Training.—All patients are referred to the Speech Therapy Department after their palates have been repaired. Four weeks after operation they are taught blowing exercises to strengthen the muscles of the soft palate and nasopharyngeal valve. The speech therapist merely guides the natural acquisition of normal speech in the small children, whose palates have been repaired early, but in the older patients who have already learnt to speak incorrectly, prolonged individual tuition is essential.

2. Orthodontic Treatment.—Whenever the cleft has involved the alveolus, the incisor teeth in the region of the cleft tend to erupt in abnormal positions or alinement. If these incisor deformities are not corrected early, lisping or other difficulties in the production of clear sibilant sounds will occur. It is important, therefore, to refer all these patients, after operation, to a dental surgeon skilled in orthodontic treatment so that the incisors can be guided during eruption into correct alinement.

3. Ear, Nose, and Throat Treatment.—A high proportion of treated and untreated cleft-palate patients suffer from Eustachian catarrh, and 15 per cent of our patients suffered from periodic attacks of middle-ear disease. Deafness may follow, and minor degrees of this may not be recognized in childhood. This interferes considerably with the development of normal speech and should be remedied as soon as possible. For this reason it is important for patients to be under the supervision of an ear, nose, and throat specialist after operation. But, in our experience, removal of adenoids with a curette is an unwise procedure after repair of the palate, because incompetence of the nasopharyngeal valve may follow. Tonsillectomy is not carried out as a routine procedure, but if the tonsils appear to be foci of recurrent infection they can, without harm, be removed by dissection.

RESULTS

An analysis has been made of 300 consecutive cases of cleft palate treated by operation during the last fifteen years. Of these cases, 205 were 'primary' and 95 were 'secondary', the latter having

had previous unsuccessful operations elsewhere; 29 patients had other associated congenital defects. There have been 5 deaths following operative treatment in the 300 cases—a mortality of 1·6 per cent.

1. Speech Results (*Table V*).—In the examination of the 183 cases that have been reviewed for speech results, the patient was asked to articulate the consonant sounds which often cause difficulty when the palate has been cleft. The results have been tabulated on a chess-board type of record card. Following conversation with the patient, nasal tone, huskiness of the voice, and the general character of the articulation were recorded in a separate column. The cases were classified into three grades :—

Grade I. Excellent. Patients with natural speech. (87 *cases.*)

Grade II. Good. Patients whose speech was greatly improved, and who could speak so clearly that strangers could easily understand them. But on careful investigation it was found, even though most of the individual consonant sounds could be pronounced correctly, they still betrayed evidence of nasal intonation or huskiness of the voice when excited or talking fast. In certain cases the sibilant sounds, although pronounced, were imperfect. (79 *cases.*)

Grade III. Bad. Patients whose speech had all the typical cleft-palate characteristics and who were difficult for strangers to understand. (17 *cases.*)

Favourable speech results, i.e., Grades I and II, were found in 90·7 per cent, and unfavourable results in 9·3 per cent. Amongst the 113 ' primary ' cases, 61 per cent were classified as ' excellent ' (Grade I), 32·8 per cent as ' good ' (Grade II), and 6·2 per cent as ' bad ' (Grade III). Amongst the 70 ' secondary ' cases only 25·7 per cent were ' excellent ', 60 per cent were ' good ', and 14·3 per cent ' bad '.

Table V.—Speech Results in 183 Cases of Cleft Palate submitted to Operation
(Primary cases, 113; Secondary cases, 70)

	Excellent		Good		Bad	
	No. of Cases	*Per-centage*	*No. of Cases*	*Per-centage*	*No. of Cases*	*Per-centage*
Primary	69	61·0	37	32·8	7	6·2
Secondary	18	25·7	42	60·0	10	14·3
Total	87	47·5	79	43·2	17	9·3

2. Anatomical Results (*Table VI*)—The anatomical results were reviewed in 183 patients. These results are considered to be of minor importance in comparison with speech, and may be summarized briefly into three groups :—

Group I : Hard and soft palate repair complete, the soft palate being muscular, mobile, and arching well. Nasopharyngeal valve competent. (163 *cases.*)

Group II : Soft palate repaired by Gillies-Fry procedure with obturator in hard-palate defect. Nasopharyngeal valve competent. (9 *cases.*)

Group III : Incomplete closure, with either defects in the hard palate or the soft palate being immobile and stiff. Nasopharyngeal valve incompetent. (11 *cases.*)

In 94·7 per cent of the ' primary ' cases, closure was complete, the soft palate arched well, and the nasopharyngeal valve was competent; 1·76 per cent were in Group II, and 3·54 per cent were bad results and in Group III.

In only 80 per cent of the ' secondary ' cases, closure was complete, the soft palate arched well, and the nasopharyngeal valve was competent, whereas 10 per cent were in Group II and 10 per cent in Group III.

Table VI.—Anatomical Results in 183 Cases of Cleft Palate submitted to Operation
(Primary cases, 113; Secondary cases, 70)

	Group I		Group II		Group III	
	No. of Cases	*Per-centage*	*No. of Cases*	*Per-centage*	*No. of Cases*	*Per-centage*
Primary	107	94·7	2	1·76	4	3·54
Secondary	56	80·0	7	10·0	7	10·0
Total	163	89·1	9	4·9	11	6·0

SUMMARY

1. The levator palati sling which arches the middle third of the soft palate, and the ' horse-shoe ' muscle-fibres which raise Passavant's ridge, are the most important agents effecting the nasopharyngeal occlusion necessary for consonant formation. Repair and reinforcement of the levator sling is therefore essential in repair of a cleft palate.

2. It is more important to provide a rapidly arching soft palate than a long and flaccid one. Length is not so important as muscularity and mobility.

3. Isolated cleft palate of Veau Types I and II, when inherited, are probably carried in a different gene complex from Types III and IV. Types I and II are more common in girls, and in these there is often a deficiency of soft tissue and sometimes they are associated with deficiency of the mandible. Types III and IV are more common in boys and there is probably no deficiency of soft tissue, but merely failure of fusion.

4. It is considered that the best results follow repair of a cleft palate by an operation based upon the principles of Veau as elaborated by Wardill and Kilner. The posterior palatine arteries should never be divided. The communicating artery between the anterior and posterior palatines should be divided between ligatures. Lateral packs should not be left in place after operation.

5. Pharyngoplasty should not be associated with a primary repair of the palate. Occasionally, however, in secondary cases a pharyngoplasty by Wardill's method may be indicated.

6. Hare-lip and cleft-palate deformities should be notified at birth to an appropriate regional authority, as in Scandinavia, so that proper arrangements can be offered for the repair of these deformities in special centres at the correct age and so that speech training can be arranged afterwards.

I should like to thank my anæsthetists, Dr. F. G. Mackintosh and Dr. R. L. H. Harris. I am indebted to my registrars, Mr. W. N. Wild and Mr. J. S. P. Wilson, for their assistance in the 'follow-up' of patients. I am grateful to my colleagues on the staff and the governing boards of the General Infirmary at Leeds and St. James's Hospital for all the facilities that have been provided for this work.

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CONCERNING THE UNFAVOURABLE LATE RESULTS OF CERTAIN OPERATIONS PERFORMED IN THE TREATMENT OF CARDIOSPASM

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THIS report is based upon a follow-up of 25 patients who complained originally of cardiospasm and who were treated, after medical methods had proved of no avail, by œsophagogastrostomy or cardioplasty. It is our purpose to show that these operations, which have been heralded with enthusiasm by others, often produce harmful effects more serious to the patients than the original lesions for which they were performed. The term 'cardiospasm' is here used to describe a condition in which there is an obstruction to the passage of food from the œsophagus to the stomach without an organic stricture; it is conveniently applied to a localized narrowing of the œsophagus which is generally idiopathic but which may be associated with pathological processes in the abdomen or the nervous system. Whatever the cause or the nature of this abnormality the differences between cardiospasm and organic stricture are sharply defined, and we can assert that at the time of operation the patients under consideration were not suffering from stricture or œsophagitis, but were in fact true cases of cardiospasm. This point is most relevant to our argument because untreated cardiospasm is not complicated by 'œsophagitis'.

The details of operative technique will not be described; we have followed the accepted methods and believe that the success or failure of these operations is concerned with other factors. *Nineteen of the patients were submitted to œsophagogastrostomy* (N. R. B.) and in these the fundus of the stomach was brought up through the diaphragm and anastomosed to the side of the œsophagus about 1½ in. above the cardia, and *in 6 patients a cardioplasty was performed* (R. H. F.), by dividing the œsophagogastric junction in a longitudinal direction through all coats and suturing the resulting incisions transversely. The first series does not include any patient in whom a Heller's operation was done, and we distinguish this procedure, which is an extramucous œsophago-cardiomyotomy (first performed by Heller, of Leipzig, on April 14, 1913) from cardioplasty as defined above; we believe that Heller's operation is a sound procedure whereas the two operations at present under consideration are unsound.

The operations were easy to perform, especially as the cardia was practically always approached by thoracotomy; there was no operative mortality and post-operative convalescence was smooth. The anastomoses were done in a variety of ways, using all types of interrupted and continuous suture materials, and exactly the same principles cover œsophagogastric anastomoses as apply to anastomoses in other parts of the gut. Irrespective of the suture material, or of the technique of stitching, the healing is rapid and satisfactory provided tension is avoided. It has been stated by other surgeons that the tendency for the stoma to contract, which may occur after any anastomosis involving the œsophagus, can be counteracted by using interrupted stitches of thread or silk; this is not our experience and when contraction does occur it is due to another factor.

To sum up, we believe that the late harmful effects of anastomotic operations, done to relieve achalasia, are not due to the way the anastomoses have been performed but are due to the fact that

an abnormal communication has been created between the stomach and the œsophagus.

THE EARLY RESULTS OF ŒSOPHAGO-GASTROSTOMY AND CARDIOPLASTY

The immediate relief afforded by these operations is striking and few patients are more satisfied than those whose dysphagia has been totally relieved. The peculiar difficulties under which people suffering from cardiospasm exist are not well enough appreciated. Their disability, if it be severe, imposes upon them a life hemmed in by intolerable limitations; many are forced to take their meals alone; special foods must be secured and prepared; they cannot eat in other people's houses or in restaurants; holidays are impossible, and married life is often severely strained. All these evils are immediately swept away by either type of operation, and as soon as they leave the hospital the patients often elect to go away for their first holiday for many years. Nor is dysphagia the only symptom to be relieved; regurgitation and retrosternal pain are abolished, appetite improves, weight increases, and anæmia is remedied (points which are influenced not only by the amount of food and vitamins which reach the intestines but by the free access of saliva to the stomach). It is our belief that this anæmia is a starvation phenomenon and is not comparable with that which may occur after operation.

The symptoms described above are the most dramatic; there are others which are also important but not so generally recognized. Long-standing cardiospasm may be complicated by persistent pyrexia—due to stagnation of food and saliva in the gullet—by severe and crippling osteo-arthropathy, and by pulmonary complications—due to the fact that the œsophageal contents spill over into the lungs when the patient lies down at night. These last symptoms are *permanently* relieved by any operation which creates a free passage between the œsophagus and the stomach or the intestine.

The hey-day which follows anastomotic operations does not usually last more than three to six months, and although some patients—contrasting their present existence with that which obtained before operation—are satisfied, they are abnormal in one or more respects and some become dangerously ill.

THE LATE RESULTS OF ŒSOPHAGO-GASTROSTOMY AND CARDIOPLASTY

Amongst the 19 patients who underwent œsophagogastrostomy 3 state that their symptoms have been relieved and that they are clinically well at present, but all the others are suffering from defects which range from trivial to serious. A follow-up of the cardioplasty patients shows that 3 are symptomatically cured, but 4 have evidence of œsophagitis; of the 6 patients only 1 has no signs or symptoms on careful investigation.

The causes of failure in both these operations are similar and will be considered together. Two things go wrong. First, some of the patients develop a new type of dysphagia after the initial post-operative period of freedom. It is most marked after œsophagogastrostomy and is partly due to kinking at the anastomosis and partly to contraction of the stoma. The latter seems to occur to some extent irrespective of the size of the aperture created at the operation and quite irrespective of the suture material used. The resultant dysphagia is never as bad as before operation and the patients generally manage to overcome it either by taking effervescent drinks with their meals or by some trick of contraction of the abdominal muscles or of the muscles of respiration. Dysphagia can also occur as a result of spasm and the radiological appearances after a barium swallow may be difficult to correlate with the patient's symptoms. One patient who had had severe hæmorrhages following an operation for cardiospasm expressed herself as completely relieved as regards any difficulty in swallowing, and yet there was a delay in emptying of the œsophagus of several hours. In another, who has recently had a Heller's operation, and who " can now swallow anything without any difficulty ", the radiographs before and after operation are practically identical.

However, dysphagia is not the serious problem; it is the almost constant development of ' œsophagitis ' which, in our opinion, puts these operations out of court. ' Œsophagitis ' results inevitably from any operation which establishes a new anastomosis between the stomach and the œsophagus and so destroys or short-circuits the normal ' sphincter mechanism ' at the lower end of the œsophagus.

At this point it is necessary to define ' œsophagitis ' and to dispel the confusion which is commonly associated with the use of this term. Apart from specific inflammations due to trauma, tuberculosis, syphilis, etc., and excluding inflammations which reach the gullet from without, four different types of ' œsophagitis ' have been observed :—

The first was described by Allison and is an inflammation due to *regurgitation of gastric contents from the stomach into the lower œsophagus*; it occurs as a result of temporary or permanent incompetence of the cardia and the changes induced are due to digestion of the œsophagus; they are similar to those which develop in the skin of the abdominal wall around a jejunal fistula. The œsophagus is digested by the gastric secretions, the mucosa is at first in a state of congestion and inflammation and this is apt to proceed to ulceration; the condition has been referred to by Allison as ' peptic ulceration '. It is this type of ' œsophagitis ' which complicates anastomotic operations and with which we are now concerned.

The second is ' *peptic ulceration of the œsophagus* ' *occurring in an islet of ectopic gastric mucosa.* This condition is often wrongly considered to be the same as the ' peptic ulceration ' described above; it has nothing to do with regurgitation of acid pepsin from the stomach. A chronic peptic ulcer, similar to that which may be found in a Meckel's diverticulum, is formed, and this occurs at the top of the œsophagus as often as at the lower end. Such ulcers are rarely seen and are of little practical importance.

The third variety can be called ' *acute ulceration* ', and in our experience may complicate any operation upon the œsophagus in which both vagi have been divided. The ulcers are similar to *acute* peptic

ulcers of the stomach or the duodenum; they come suddenly out of the blue and may heal spontaneously or become chronic; they may bleed or perforate. The type of operation which can be complicated in this way is excision of the lower œsophagus for carcinoma with anastomosis in the mediastinum

secretions; in each of these patients both vagi had been taken out with the tumour.

The fourth type is best described as '*retention ulceration*' and is relevant to this discussion because it is often found in advanced cases of cardiospasm *before operation*; it is due to inflammation of

LATE RESULTS OF ŒSOPHAGOGASTROSTOMY AND CARDIOPLASTY FOR CARDIOSPASM

CASE	DYSPHAGIA	ANÆMIA	PAIN	BLEEDING, i.e., HÆMATEMESIS OR GROSS MÆLENA	SYMPTOMATICALLY CURED	REMARKS
1	—	+	—	—	—	
2	—	+	—	—	—	—
3	+	+	+	+	—	Dead
4	+	+	+	—	—	—
5	+	—	—	—	—	—
6	—	+	—	—	—	—
7	—	—	—	—	Cured	—
8	+	—	—	—	—	—
9	+	—	—	—	—	—
10	+	+	+	+	—	—
11	—	+	—	—	—	—
12	—	+	—	—	—	—
13	—	+	—	—	—	—
14	—	—	—	—	Cured	—
15	—	+	+	—	—	—
16	+	+	+	—	—	—
17	—	—	—	—	Cured	—
18	—	+	—	—	—	—
19	+	—	+	—	—	—
19 cases	8 dysphagia	12 anæmia	6 pain	—	3 cured; 16 have evidence of œsophagitis	
1	—	—	—	—	+	Transthoracic cardioplasty
2	—	—	—	+	+	Transthoracic cardioplasty. Occult blood in stool before and after operation
3	—	+	+	+	—	Heller's operation stated to have been carried out previously. Later transthoracic cardioplasty. Œsophagitis seen with œsophagoscope
4	—	+	—	—	+	Transthoracic cardioplasty
5	+	—	+	+	—	Abdominal cardioplasty
6	+	+	+	+	—	Heller's operation said to have been carried out without improvement. Later transthoracic cardioplasty. Repeated severe hæmorrhages
6 cases	Three symptomatically cured—4 have evidence of œsophagitis: *Case* 6 had a small hæmorrhage before any operative treatment was undertaken and is possibly a case of œsophagitis made worse by the procedure.					

between the proximal part of the gullet and the stomach. During the last year this complication has occurred five times in our practice (N. R. B., 4; and R. H. F., 1), and it cannot be ascribed to regurgitation of acid pepsin, for in carcinoma cases there is seldom free hydrochloric acid in the gastric mucosa caused by stagnation of food and saliva above the block. It is analagous to 'stercoral ulceration' in the lower bowel, and has no relationship to regurgitation. Retention ulceration is a cause of persistent pyrexia, and is relieved by *any* operation which overcomes œsophageal obstruction.

THE CAUSE OF ŒSOPHAGITIS AFTER ŒSOPHAGOGASTROSTOMY AND CARDIOPLASTY

The cause of œsophagitis in the cases under consideration is incompetence of the cardia induced either as a result of short-circuiting the cardia, putting it out of action by a cardioplasty, or by performing any operation which leaves the œsophagogastric junction above the diaphragm. It is a matter of practical experience that Heller's operation does none of these things, whereas both the operations we are considering set the stage for regurgitation.

The nature of the mechanism which normally prevents regurgitation from the stomach into the œsophagus has been the subject of much speculation and investigation and the only point of common agreement is that it is one of extreme complexity. A few facts are substantiated; any arrangement which results in the cardia lying above the diaphragm may lead to incompetence, but the converse is not true; for instance, if the œsophagus be brought through an artificial hole in the diaphragm and its natural hiatus be closed (as it may be in the repair of some types of diaphragmatic hernia) the cardia becomes incompetent although it lies below the diaphragm—the explanation of this may be that the œsophagus, in its new position, enters the stomach as a tube enters a funnel, and not obliquely into the inner side of a curve as it does in its normal position. Allison has drawn attention to the fact that if the stomach and œsophagus are removed from the cadaver and laid out flat, distension of the stomach with liquid can be carried out without regurgitation into the œsophagus owing to the angle at which the latter enters the stomach and the valve effect produced by distension of the fundus upon this angle. The mechanism is aided in life by the decussating fibres of the right crus which form the œsophageal hiatus and by the anatomy of the 'liver tunnel'. The fact is that although no muscular sphincter can be demonstrated at the cardia the anatomy of the parts is such that regurgitation does not normally occur, and it is easy to see that the operations we have performed upset this anatomy whereas Heller's operation does not. The matter is complicated by the knowledge that sliding of the cardia from its anatomical position into the thorax occurs normally at the extremes of life and without ill effect, but it may be that in both these states the amount of acid pepsin in the gastric secretions is below the harmful level. It is certain that adults who suffer from 'heartburn' occasionally, are experiencing transient attacks of incompetence of the cardia, and the normal mechanism is upset in some cases of debility, in certain types of diaphragmatic hernia (but not in that variety of para-œsophageal hernia in which the cardia is in its normal place), and whenever a person vomits.

The extreme susceptibility of the œsophageal mucosa to the digestive effects of gastric juice was shown in the following case :—

A small boy was admitted to Horton War Hospital under one of us (N. R. B.) suffering from a 'stricture of the lower œsophagus' which had resulted from œsophagitis occasioned by long-standing incompetence of the cardia. The stricture was in fact a protective mechanism which nature had set to prevent further regurgitation, but at the time the true significance of the stenosis was not understood. Attempts were made to dilate the stricture. Œsophagoscopy was performed and it was observed that the mucosa of the œsophagus was normal as far as the stricture; the latter was carefully dilated to such an extent that a small laminaria tent could be introduced. Twenty-four hours later the laminaria tent was removed and it was again noticed that there was no evidence of inflammation or indeed of any abnormality in the mucosa above the stricture, which had been temporarily blocked up by the laminaria tent. As a result of this operation there was such a favourable improvement in the boy's ability to swallow that it was decided to introduce a large laminaria tent. Accordingly another œsophagoscopy was performed, twenty-four hours after the first laminaria tent had been taken out, and on this occasion the whole mucosa of the lower third of the œsophagus was in a state of acute hæmorrhagic ulceration and liquid could be seen welling up through the dilated stricture from the stomach.

A boy came under the care of one of us (R. H. F.) at the age of 18 months with a history that, ever since taking solid food, he had had some difficulty in swallowing. Even with fluids he often suffered from regurgitation. X-ray examination showed 'a stricture' at the point of junction of 'a short œsophagus' with the stomach, it being apparent that a portion of the stomach was lying in the thoracic cavity (*Fig.* 267). The only question of doubt was whether the condition should be regarded as a congenital short œsophagus or an example of

FIG. 267.—This film was taken on Jan. 1, 1947, when the patient was an infant. It shows that at this time the cardia was lying just above the diaphragm and there was a constriction in the lumen of the gullet at this point.

ulceration of the œsophagus followed by shortening as part of the process of healing. Treatment was undertaken by dilatation and clinically the child was improved. After discharge from the hospital, symptoms recurred and necessitated further treatment. At this time it was recognized that dilatation might open up further tracts of the œsophagus to the effects of the gastric juices, but in view of the dysphagia it was decided to continue treatment in spite of this risk

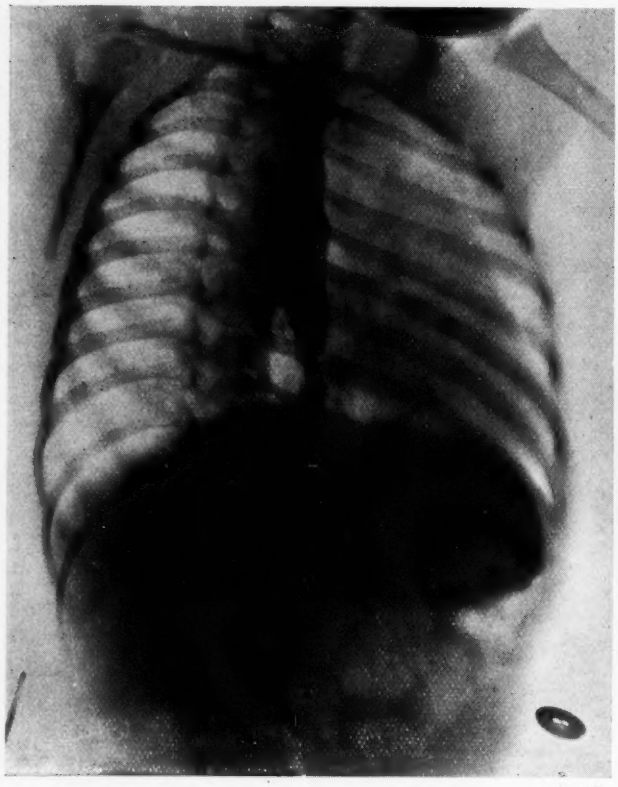

FIG. 268.—Film taken on Feb. 5, 1948. The cardia is still above the diaphragm and there is some dilatation of the œsophagus above the stricture.

FIG. 269.—Film taken on March 5, 1949. The cardia is now in its normal position and there is no clinical or X-ray evidence of œsophagitis or stricture.

(*Fig.* 268). After the last course of dilatation a further X-ray examination was carried out and showed the remarkable fact that the stomach was now lying completely in the abdomen and the œsophagus appeared of normal length (*Fig.* 269). It seems clear that this is an example showing that, at the extremes of life, the position of the cardia is subject to variations so that both œsophagitis and herniation of the stomach into the thorax are processes which can be corrected spontaneously.

THE PATHOLOGY OF ŒSOPHAGITIS IN THIS GROUP OF CASES

The lesions produced in so many of this group of patients involves the lower portion of the œsophagus—namely, that part which one might expect to be the first to suffer if regurgitation of gastric juices were in fact to be the cause. The characteristic œsophagoscopic appearance is hyperæmia of a mucosa which tends to bleed easily and to form superficial, angry-looking, ulcers. The latter may heal and break down in a way which is comparable with the cycle found in duodenal ulcers; if healing has occurred patches of leucoplakia may be seen. Allison states "the ulcers appear usually as pale yellowish-grey areas surrounded by a well-defined bright pink margin. When the fibro-purulent membrane is removed by a swab the whole surface of the ulcer shows the same bright colour as the margin and oozing of blood readily occurs." Increased vascularity and a tendency to bleed are the striking features and the condition can be compared with internal piles, which can cause hæmorrhage out of all apparent proportion to their size. Spontaneous improvement is common, and, during periods when the patients are symptom-free the appearances of the mucosa may return almost to normal. The condition has a strong tendency to relapse, and ultimately, after repeated attacks, may lead to involvement of the muscular layers of the œsophagus and to the formation of organic stenosis, peri-œsophageal adhesions, and inflammatory adenitis.

Healing may thus result in both 'shortening' of the œsophagus and stricture formation. It is our experience that the lesion remains limited to the mucosa and submucosa for a long time, and none of our patients has actually developed a full-fledged fibrous stricture, although some have suffered from œsophagitis for many years. The condition does not apparently predispose to malignant disease, although carcinoma sometimes develops in the œsophagus above a cardiospasm.

THE SYMPTOMS AND SIGNS OF ŒSOPHAGITIS IN THESE PATIENTS

The outstanding abnormalities were bleeding and pain; dysphagia, as has been explained above, was sometimes present as well. These signs and symptoms varied in severity from patient to patient, and in the same individual from time to time, and it is the variations which may account for the fact that the lesions have not already been reported by other surgeons.

The tendency of the condition to become quiescent at times is comparable with a similar lesion in the duodenum, when patients may suffer

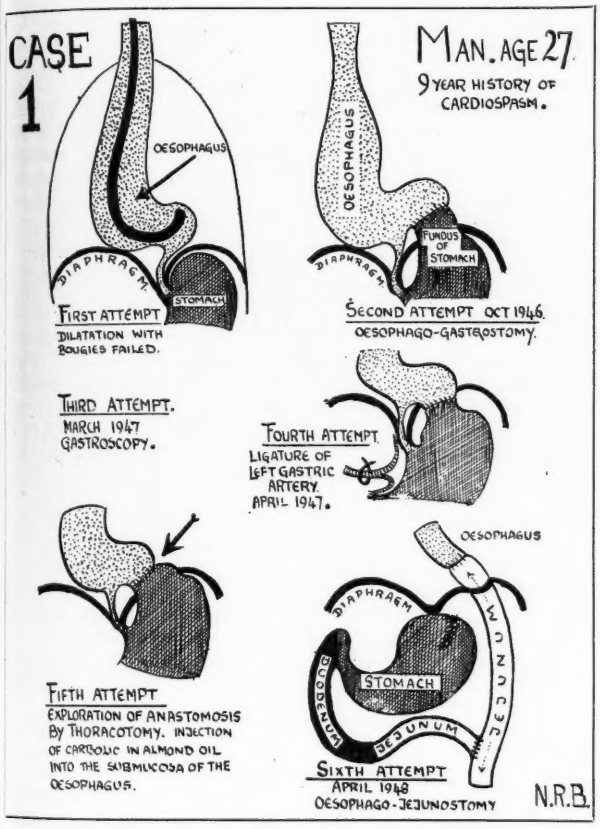

FIG. 270.—*Case* 1. Diagram showing the various procedures adopted to effect a cure.

from attacks of duodenal pain without the formation of a very obvious ulcer.

The variation in the clinical results and in the amount of the *bleeding* is extreme; in some patients anæmia is never a prominent feature whereas in others it may be very dangerous. The amount of hæmorrhage and the severity of the anæmia do not appear to depend on the extent of the ulceration which can be seen at œsophagoscopy, and may be maximal in those cases where the local lesion seems to be hyperæmia. We submit that in these circumstances avitaminosis may play a part in influencing the blood-loss. Examples will illustrate these points:—

Case 1 (*Figs.* 270, 271).—A man aged 27 was operated upon (N. R. B.) in October, 1946, for severe cardiospasm of nine years' duration. An œsophagogastrostomy was performed by thoracotomy without difficulty and within twenty-four hours he was suffering from all the signs of internal hæmorrhage. Transfusions were given and his condition improved slowly, but the stools contained occult blood and within a fortnight his hæmoglobin had been reduced to less than 30 per cent. Repeated transfusions were given, but six months later the bleeding was still going on and he was transferred to King's College Hospital under the care of Mr. Harold Edwards. The latter performed gastroscopy and reported that although no obvious abnormality could be detected at the anastomosis he felt the bleeding must be coming from a lesion high up upon the lesser curve of the stomach. In March, 1947, Harold Edwards did the second operation, a laparotomy, and reported "the findings were disappointing and the only observation I could make about which there was no question or doubt was that the lesser curve of the stomach was normal. . . . I was not able to get a clear view of the anastomosis . . . but tied the left gastric artery. . . . The anastomosis was firmly adherent to the *under*-surface of the diaphragm".

The post-operative course was uneventful and at first the bleeding stopped and the hæmoglobin rose to 90 per cent in May, 1947. Two months later he was

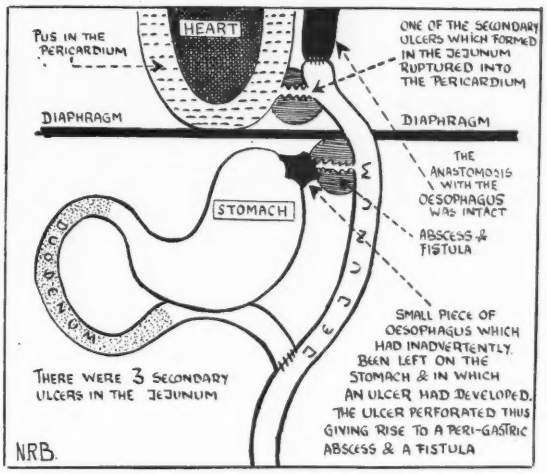
PUS IN THE PERICARDIUM
HEART
ONE OF THE SECONDARY ULCERS WHICH FORMED IN THE JEJUNUM RUPTURED INTO THE PERICARDIUM
DIAPHRAGM
DIAPHRAGM
THE ANASTOMOSIS WITH THE OESOPHAGUS WAS INTACT
STOMACH
DUODENUM
JEJUNUM
ABSCESS & FISTULA
SMALL PIECE OF OESOPHAGUS WHICH HAD INADVERTENTLY BEEN LEFT ON THE STOMACH & IN WHICH AN ULCER HAD DEVELOPED. THE ULCER PERFORATED THUS GIVING RISE TO A PERI-GASTRIC ABSCESS & A FISTULA
THERE WERE 3 SECONDARY ULCERS IN THE JEJUNUM
N.R.B.

FIG. 271.—*Case* 1. Post-mortem findings.

re-admitted to King's College Hospital in a serious state of anæmia, with much occult blood in the stools. He was given 6 pints of blood, bringing the total transfusions at this time to 68 pints.

In September, 1947, he was still bleeding and the third operation was performed by N. R. B. The anastomosis was explored through the chest; this operation was difficult, but after the parts had been identified the gut was opened and the region of the anastomosis inspected. The stoma was widely patent and was situated just below the diaphragm; no ulceration was found but the mucosa bled very easily and was red and inflamed. Some carbolic acid in almond oil was injected into the submucosa and the operation was concluded. Convalescence was again uneventful, but the bleeding continued unabated.

In February, 1948, he was again admitted to King's College Hospital with severe anæmia and persistent occult blood in the stools. The hæmoglobin was 23 per cent and the red-cell count 2,160,000. He was given 9 pints of blood. There was no abnormality in the blood itself to account for the bleeding. An œsophagoscopy was performed by Mr. Lewis, who reported that there was considerable regurgitation of gastric contents into the lower œsophagus and that there was "a large granulating area in the œsophagus extending from 28 to 34 cm.". The bleeding continued and further transfusions were given.

In April, 1948, the fourth operation was done at St. Thomas's Hospital (N. R. B.) and by this time the patient had been given over 100 transfusions. The chest was opened and after many difficulties the anastomosis was identified and freed for four-fifths of its circumference. The œsophagus was divided and the stomach closed; a loop of jejunum was prepared, after the manner described by Yudin, and this was joined by end-to-end anastomosis to the cut part of the œsophagus. The proximal part of the jejunal loop was anastomosed to the side of the distal part about 10 in. below the œsophagojejunal suture line. This operation nearly destroyed the patient, but he survived, became convalescent, and never again suffered from hæmorrhage. The lower end of the œsophagus was examined histologically and showed all the typical changes of acute œsophagitis.

In September, 1948, the patient died after a short illness, during which he was judged to have a perforated peptic ulcer of the stomach and a laparotomy was performed without benefit. Before death he developed acute pericarditis and "tubercle bacilli" were found in the liquid aspirated from the pericardium. There was no bleeding during this illness.

At autopsy it was shown that at the fourth operation a small piece of œsophageal mucosa had been left upon the stomach when the œsophagus was cut across. An ulcer had developed in this patch in the stomach and had perforated through into the jejunal loop which had been used to make the œsophagojejunal anastomosis; a gastrojejunal fistula had thus developed and through this gastric juices had gained access directly to the distal jejunal loop. In the latter there were three acute ulcers and one small one just above the œsophageal anastomosis. The upper one of these ulcers had perforated into the back of the pericardium, giving rise to suppurative pericarditis from which he died. The "tubercle bacilli" which had been found in the pericardial pus were timothy grass bacilli which are generally demonstrable in liquids removed from the œsophagus. (*Fig.* 271.)

Case 2.—A woman, aged 47, was operated upon in 1937 for advanced cardiospasm and an œsophagogastrostomy was performed. She has been seen regularly since that time and, on the whole, has been pretty well according to her own statements. In fact, she has suffered from continuous anæmia, which has varied from severe to mild and which has been treated by transfusions, a special diet of milk and alkalis, and the exhibition of iron. Barium swallows show an irregularity of the mucosa at the lower end of the œsophagus, but the stoma functions well and there is no stricture or retention. Œsophagoscopies have been done on several occasions and always show œsophagitis—sometimes ulceration is present and sometimes it is not, but always "the mucosa bleeds very easily on touching or stretching". Her hæmoglobin has never been above 65 per cent since the operation.

Case 3.—A girl, aged 10, had an œsophagogastrostomy performed in September, 1947. Her dysphagia was completely relieved and she made splendid progress until November of the same year when she began to feel and to look tired. The stools were noticed by her parents to be tarry, and this was due to blood. During the following month the symptoms were aggravated and she started vomiting 'coffee-ground' material. A barium swallow showed that the stoma was functioning well, but she had acute œsophagitis and profound anæmia. In this case the bleeding stopped after transfusions, and a year later the child was well, there was no anæmia, swallowing was normal, and the œsophagitis seems to have settled down. It is our opinion that this patient may develop further trouble in later life.

Case 4.—A case of œsophagitis diagnosed as cardiospasm and made worse by cardioplasty.

A woman aged 47.

1934: Onset of dysphagia. Subjective sensation of obstruction to passage of solid foods at the level of the suprasternal notch. No difficulty in swallowing fluids. Retrosternal pain.

1940: Regurgitation occurred for the first time.

1946: Small hæmatemesis—the occurrence of which was not ascertained until a later date. Later in 1946 Heller's operation is said to have been carried out through the abdomen. No improvement followed this operation and the passage of Hurst's mercury bougies was attempted unsuccessfully.

1947: August. Transthoracic cardioplasty (R. H. F.) Immediate result excellent.

1948: February. Recurrence of dysphagia associated with substernal pain. Spontaneous recovery after six weeks.

1948: May. Severe hæmatemesis necessitating blood transfusion. In spite of treatment with alkalis and olive oil and the adoption of a raised position for sleeping, this patient has since suffered from another considerable hæmorrhage. Symptomatically her swallowing is satisfactory and her weight has been maintained, but the barium swallow shows a considerable delay and she still gets attacks of pain, especially at night and if she lies on the right side. The correct diagnosis was made difficult by the distortion produced by the first operation and by the fact that œsophagoscopy was never entirely satisfactory. X-ray examination on no occasion showed any evidence of gastric or duodenal ulcer. This patient would appear to have suffered from œsophagitis from the start and the operation of cardioplasty improved her swallowing at the expense of the mechanism preventing regurgitation into her œsophagus, and so led eventually to an œsophagitis of increased severity.

Case 5.—A woman, aged 54, gave the history that eighteen months previously she had suddenly found difficulty in swallowing solids. The dysphagia was accompanied by epigastric pain which travelled up to the neck and which was relieved by taking more food or more so by drinking cold water. She has lost 4 st. in weight since the onset of her symptoms. X-ray examination showed the typical appearance of achalasia, with marked dilatation of the œsophagus.

Feb. 28, 1947: Cardioplasty by the abdominal approach (R. H. F.). The immediate subjective result was excellent. X-ray examination still showed dilatation of the œsophagus, but the hydrostatic pressure required to overcome the spasm was less than before operation. Three months later she had gained 1 st. in weight, but was noticing that her food seemed to stick from time to time.

Eighteen months after the operation she started to regurgitate altered blood.

It is to be noted that, although from the specimens which this patient brought for inspection there had evidently been quite a considerable amount of 'coffee-ground' material regurgitated, her general condition remained good and she expressed herself satisfied with the result of the operation.

A search of the literature has not revealed similar records of anæmia occurring after œsophagogastrostomy or cardioplasty, but there are many isolated cases described in which bleeding has been noticed as a complication of some types of diaphragmatic hernia. It is noteworthy that in such the bleeding has been ascribed to the fact that the veins draining the submucosa of the herniated part of the stomach were congested on account of pressure at the hernial orifice and that the hæmorrhage was due to the venous engorgement which resulted. Anæmia

in patients suffering from diaphragmatic hernia is much more likely to be due to œsophagitis or gastritis than to mechanical effects causing venous obstruction.

The other prominent symptom is *pain* and this may be associated with bleeding in some cases or it may be the only abnormality. It is common to see patients who have œsophagitis, the presence of which has been proved by endoscopy, who have no pain.

There are several different types of pain and thoracic discomfort associated with cardiospasm and with the pathological conditions which occur after some operations. In the early stages of cardiospasm, when peristalsis and irritability of the œsophagus are at their height, the patient often complains of cramp-like pains in the thorax which are due to abnormal peristalsis and are connected with attempts to swallow solid or liquid foods. Later on, when the œsophagus is permanently distended and acting as a stagnant reservoir for food and saliva, the patient complains of a feeling of heaviness in the base of the right chest and if ' retention ulceration ' be present there may actually be a feeling of soreness and stiffness referred to the epigastrium and the back of the thorax. When an operation has been performed and œsophagitis has come on, a new pain appears; this is described as ' heartburn ' and is situated behind the xiphisternum and in the upper epigastrium. It is generally induced by taking food, but may be relieved in this way. The pain can be severe and may radiate through to the back, down the arm, or up the neck to the jaw. The type of pain which radiates is apt to be wrongly ascribed to cardiac or gall-bladder lesions. One cause of the pain is the presence of free hydrochloric acid impingeing upon an œsophageal mucosa which has already been partly digested; in proof of this we quote the following simple experiments. A number of normal and abnormal people were given 2–4 oz. of 0·3 per cent hydrochloric acid to drink slowly over a period of five minutes. Those who had never had indigestion suffered no ill effect; those who occasionally experienced ' heartburn ' got a typical episode of this condition, as did a small group of patients who were known to have œsophagitis. Those who got heartburn experienced no pain if normal saline was taken, and the pain could be straightway alleviated by giving alkalis.

THE LESSONS LEARNT FROM CARDIOPLASTY AND ŒSOPHAGOGASTROSTOMY

As we have abandoned cardioplasty and œsophagogastrostomy because these operations produce incompetence of the cardia and œsophagitis, we believe that in the future our patients will not suffer from these disabilities. In the process of coming to this conclusion certain observations have been made and certain lessons learnt.

Patients presenting the signs and symptoms of *cardiospasm* must be thoroughly assessed before treatment if mistakes are to be avoided. Complete diagnosis is not always possible, but in every case an œsophagoscopy is an essential investigation. Conservative treatment consists of efficient dilatation of the constricted segment of the œsophagus, using—for preference—Negus's hydrostatic dilator. Some of the mildest cases may respond to antispasmodic drugs and to suitable regulations of the diet; but the swallowing of mercury bougies is, or should be, a thing of the past. If operation is found to be necessary nothing which interferes with the competence of the cardia must be done, and the success which follows Heller's procedure rests upon this cardinal point.

In the case of children, in particular, there should be no question of undertaking an operation lightly, especially in view of the case which has been quoted above in which the stomach spontaneously assumed a normal position after the stricture had been dilated over a period of nearly two years.

Most of the patients at present under consideration have lived for months or years with varying degrees of disability. The majority no longer complain of dysphagia and have maintained a fairly level subnormal standard of health; but they have suffered from anæmia or pain. These symptoms have waxed and waned for unaccountable reasons, and in some cases have shown long periods of absolute quiescence. The pain, which resembles ' heartburn ', could sometimes be relieved by taking alkali powders and olive oil at regular intervals and by sleeping sitting-up, thus overcoming the effects of gravity to some extent. One patient passed a Ryle's tube every night before retiring and connected this to a drip feed of milk at the head of the bed and was enabled to sleep without discomfort.

There were many surprising things about the anæmia. The most serious cases were often those in which the œsophagitis seemed to have produced nothing more than hyperæmia of the mucosa; in such the bleeding would be started by merely passing an œsophagoscope to the vicinity of the lesion and so stretching the mucosa in advance of the beak of the instrument. In no case was a single vessel, which could have been ligated surgically, found, and in none did the bleeding come from an eroded vessel in the floor of a chronic ulcer. The bleeding did not usually produce either serious hæmatemesis or obvious melæna, but occult blood was a regular finding in the stools; the red cells were often reduced to less than 3,000,000 and the hæmoglobin to less than 30 per cent. Mild cases responded clinically to conservative treatment and the exhibition of iron or of liver extract, but serious cases required repeated large transfusions. It follows from these observations that any operation designed to stop the bleeding by ligating local or adjacent vessels will not succeed. The only way to stop serious bleeding is to prevent gastric juices reaching the lower œsophagus.

In some patients the possibility of having to operate again was considered, and in some second and third operations were done. From these we have learnt that neither pain nor anæmia will be controlled by gastrostomy, jejunostomy, vagotomy, ligature of the left gastric artery, or injection of sclerosing fluid in the submucosa of the affected area of the œsophagus.

It was found that if the anastomosis could be isolated, unpicked, and the parts restored to normal, so that a Heller's operation could then be done and

the cardia fixed below the diaphragm, the result was good in every respect. On two occasions when such an operation was being performed, the body of the stomach together with some omentum was seen to have pushed its way up through the aperture which had been made in the diaphragm to do the original œsophagogastrostomy; thus underlining the danger of ever bringing any of the abdominal contents into the thorax through an artificial hiatus in the diaphragm. Through such a hole an ingravescent hernia will certainly occur whether the selected piece of gut or omentum be carefully stitched to the margins or not.

If the original operation cannot be converted to a Heller's procedure, there are two alternative methods of dealing with the situation. The acid gastric secretions can be removed or abolished by gastro-enterostomy, or partial gastrectomy, as recommended by Wangensteen, of Minneapolis, in some cases of œsophagitis. This method was adopted in *Case* 6. Alternatively the operation recommended by Allison may be practised, and this has been done with complete success on two occasions. The œsophagus is divided just above the zone of œsophagitis and *the whole of the affected area of the gullet is removed* down to the stomach; the stomach is then closed and the proximal end of the œsophagus is anastomosed to the small intestine. The œsophagus is thus separated completely from the stomach and swallowed food passes straight into the jejunum.

Case 6.—**Œsophagitis in association with diaphragmatic hernia.**

A woman of 54 complained of dysphagia following the repair of a diaphragmatic hernia six weeks previously (1944). The hernia had been repaired from below with catgut sutures. Treatment was started with Hurst's mercury bougies. Six months later the dysphagia was still present and, in addition, she complained of pain behind the sternum as soon as she swallowed anything.

January, 1945: Gastrostomy carried out (R. H. F.) because the patient was unsuitable for any major operation. This made no difference to her retrosternal pain and in addition she developed a very painful shallow ulcer around the gastrostomy tube.

July, 1945: Ulcer excised and gastrostomy closed (R. H. F.).

August, 1946: Gastrostomy carried out elsewhere as an emergency because of almost complete dysphagia. This did not produce freedom from pain and in June, 1947, it was replaced by a jejunostomy (R. H. F.), and further investigations were undertaken which showed that there was still a small herniation of the stomach into the thorax.

August, 1947: A transthoracic repair of the hernia was undertaken (R. H. F.) and the patient was discharged apparently greatly improved.

It transpired later that in November, 1947, she had again developed almost complete dysphagia and had suffered yet another gastrostomy in December, 1947. With this she remained in tolerable comfort until August, 1948, when the gastrostomy started leaking and there was evidence of pylorospasm which was presumably a recrudescence of duodenal ulceration from which she had suffered in the past and which had led to perforation in 1938 and again in 1939.

Œsophagoscopic examination in August, 1948, showed multiple superficial ulcers at the lower end. These ulcers were covered with slough. Attempts were made to improve the condition by alkalinization, posture, and a continuous milk drip without avail, and finally operation was undertaken (R. H. F.).

The lowest 2 in. of the œsophagus and the whole stomach were removed and continuity restored by bringing up the small bowel into the chest.

The final result has proved satisfactory.

SUMMARY

A series of 25 patients who have undergone operations for cardiospasm has been followed up. The operation consisted of either œsophagogastrostomy or cardioplasty. Some illustrative cases have also been described.

The purpose of this paper has been to show that certain procedures which alter the anatomy at the lower end of the œsophagus, including the two operations which have been discussed in particular, may result in the complete disorganization of the normal mechanism which prevents regurgitation from the stomach into the œsophagus. It has been shown by a follow-up of these cases that great care must be exercised in selecting the type of operation required in these cases in order to avoid leaving the patient ultimately in a worse position than he was originally.

RECONSTRUCTION OF THE TRACHEA AND CERVICAL ŒSOPHAGUS

PRELIMINARY REPORT

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The literature relevant to the surgery of the trachea contains many articles on tracheotomy; a fair number deal with the treatment of stenosis and fistulæ (Koenig, 1896; Levit, 1912; Neuhof, 1917); piecemeal endoscopic removal of a primary tumour has been performed many times (Clagett, Grindlay, and Moersch, 1948); resection has been recorded as a treatment for cancer of the cervical trachea; but very few papers (Belsey, 1946; Clagett, and others, 1948; Daniel, 1948; Fairchild, 1927) discuss reconstruction after resection.

The problems to be solved in reconstruction of the trachea are interesting; but opportunities for putting one's theories to clinical trial are rare. During the last thirty years only 5 patients were admitted to St. Thomas's Hospital with a malignant tumour suitable for resection and reconstruction of the trachea. Two of these patients had a primary tracheal carcinoma and three a thyroid carcinoma which had invaded the trachea but was otherwise operable.

Resection and reconstruction of the trachea is only possible if at least one recurrent laryngeal and one superior laryngeal nerve can be preserved. Bilateral recurrent laryngeal nerve palsy prevents tracheal reconstruction by obstructing the airway,

and paralysis of both superior laryngeal nerves anæsthetizes the entrance to the larynx so that the patient may inhale food into the remade trachea with fatal results.

The ideal method of reconstruction is by primary end-to-end anastomosis. That the trachea heals well after incision and suture is shown by two patients in whom incisions about 5 cm. long were made through its wall and sutured again without any post-operative complications. Unfortunately in man end-to-end anastomosis is seldom possible if a resection has been necessary. Clagett (1948) and his colleagues from the Mayo Clinic report that the trachea of dogs possesses great elasticity and that end-to-end suture is possible after the excision of a quarter of its length; this, however, is not so in man. Six fresh human bodies have been dissected and in our opinion end-to-end suture is impossible if more than 2 cm. is removed; beyond this, tension even after maximum mobilization would be too great. In the majority of patients reconstruction can only be achieved by a method which bridges the gap.

The gap which remains after resection of the trachea may be closed in a variety of ways. The experience of others with the closure of tracheal fistulæ is of help. Koenig in 1896 successfully closed a fistula by a two-stage operation. At the first stage he grafted a small piece of rib into the subcutaneous tissues of the neck; six weeks later a skin flap containing this rib was turned across the fistula. In 1911 Hohmeier, after animal experiments, closed a small defect 0·75 cm. square with a free graft of fascia lata. This work has been repeated, and Neuhof (1917) found that a patch of fascia placed over a window in a dog's trachea 0·25 cm. square was completely covered by columnar ciliated epithelium in six weeks. Others (Daniel, 1948) have used a fibrin film and the connective tissues of the neck to cover small tracheal windows in dogs.

The rebuilding of a new segment of trachea appears to be an unusual operation. In 1927 Fairchild remade a segment of the cervical trachea with a skin tube. A three-stage operation was necessary in this patient, who had a complete traumatic rupture of the trachea with considerable retraction of the ends. In addition Belsey (1946) has reported a successful reconstruction after excision of a cylindroma. And end-to-end anastomosis of a bronchus or the lower trachea has been performed several times, but not after the resection of a malignant tumour.

In dogs metallic tubes and polythene tubes have been used with success (Clagett and others, 1948; Daniel, 1948); polythene appears to be better than metal because it does not wet and therefore mucus cannot stick to it. This work with polythene tubes (Clagett and others, 1948) is of interest because in experimental animals a complete layer of epithelium rapidly grew across the gap, but this layer was outside the polythene tube and attempts to remove the tube during the life of the animal were usually unsuccessful. This is an important observation relevant to the reconstruction of any fixed body tube, because if a rigid wall is not necessary a temporary tube may be used to protect the tissue planes against infection and until epithelium has grown across the defect. If, however, a rigid wall is needed (as it is with the trachea) a tube should be used which allows the epithelium to grow on its inner side so that a normal lining membrane is restored.

In view of these findings two types of operation have been practised, one for the trachea and another for the cervical œsophagus. We are reporting the results in 4 patients. We must apologise for this small number, but suitable patients are rare.

In one patient the cervical trachea has been reconstructed. A thyroid carcinoma of low malignancy had been excised seven years before a local recurrence, which was radio-resistant, had invaded

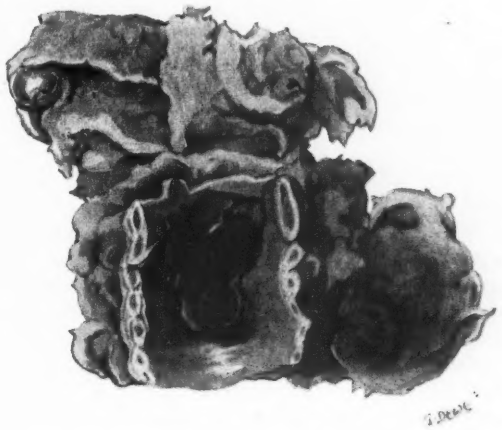

FIG. 272.—Operation specimen. Invasion of the trachea by thyroid carcinoma. The tissue removed includes a portion of the cricoid cartilage and six tracheal rings.

the trachea. He complained of hæmoptysis and respiratory obstruction. Endoscopy showed a bleeding mass on the anterior tracheal wall just below the glottis. No detail could be determined because of the bleeding. In August, 1948, the tumour was resected. This necessitated the removal of a portion of the cricoid cartilage and six tracheal rings (*Fig.* 272). It was found possible to preserve a narrow strip of epithelium on the posterior tracheal wall less than 0·5 cm. wide, both recurrent laryngeal nerves, and at least one parathyroid gland. The defect in the trachea was 9 cm. long. A primary closure was carried out using a tube of tantalum gauze covered on both sides by fascia lata (*Fig.* 273). This was sewn to the trachea with interrupted silk sutures. The skin wound was closed with a separate stab drain.

The post-operative course was interesting. During the first day he breathed satisfactorily, was able to cough up his sputum, spoke well, and had no difficulty with swallowing; he had, however, some surgical emphysema in his neck. On the second day he developed an atelectasis of the lower lobe of the right lung and at the same time found coughing difficult. Bronchial aspiration was therefore performed three times during the next 36 hours. On the third occasion a bronchoscope was passed down the trachea right through the graft; the fascia was healthy in appearance but the inner layer was seen to be drawn into the trachea with each inspiration, producing a partial obstruction.

During the next fourteen days his condition steadily improved and apart from some inspiratory stridor, presumably due to tenting inwards of the inner layer of fascia lata, he did not complain. On the sixteenth post-operative day a further bronchoscopy showed that the inner layer of fascia was still

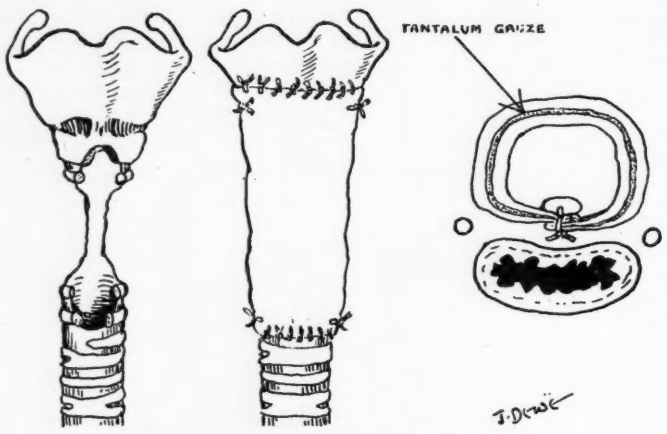

FIG. 273.—Reconstruction of the trachea.

partially occluding the lumen; it was therefore destroyed with a diathermy electrode. When the fascia was touched with the electrode it shrivelled and became adherent to the tantalum. By touching it in several places the lumen of the trachea was restored. His breathing then became free and he had no stridor. *Fig.* 274 is a radiograph showing the position of the tantalum gauze in his neck.

On the forty-third post-operative day another bronchoscopy showed an oval area of granulation tissue near the middle of the defect about 0·5 sq. cm. in size; the remainder was covered by tracheal epithelium. Next day he was discharged from hospital and three weeks later he resumed his work as a clerk. He has remained well since. A bronchoscopy performed four months after operation showed a small polyp anteriorly and just below the vocal cords, which was removed. Microscopy showed it to be granulation tissue. Apart from this the epithelium was normal and the tantalum gauze could not be seen. It is now one year since his operation and he is very satisfied. He has no symptoms and no signs of recurrence.

The interest of this patient lies in the fact that a rigid tube has been provided by the tantalum gauze and that the tracheal epithelium has grown *on its inner side*, the tantalum remaining in situ. In our opinion the reason why the epithelium grows on the inner side of such a tube but on the outer side of a polythene tube is because it is not an impervious cylinder but consists of a fine wire mesh. Granulation tissue is able to pass through this mesh, and so a granulating layer is available on the inner side of the wire on which the tracheal epithelium can grow. The fascia lata does not survive: it merely provides a temporary seal until the tissue planes of the neck have become closed around the tantalum gauze.

In three patients the hypopharynx and upper œsophagus have been resected for carcinoma, and an immediate one-stage reconstruction performed. The technique used in the third patient will be described because this was modified by lessons learnt from the first two. He had a carcinoma of the hypopharynx and upper œsophagus surrounding it except for the right lateral wall. Apart from a left recurrent laryngeal nerve palsy there was no evidence of local invasion or metastases. Through a high collar incision the carcinoma was resected together with a certain amount of fascia and lymphatic tissues.

Reconstruction was carried out with a tube of tantalum gauze covered on the outside by fascia lata. *Fig.* 275 shows the position of this tube, which was placed within the lumen of the gullet and sutured loosely at the top and bottom to its wall. This was because we intended to remove the tantalum gauze during the third week. A large-bore plastic feeding tube was passed through the nose to the mid-œsophagus and the wound closed with a small rubber drain.

The post-operative course was uneventful; the wound healed by first intention, an adequate food intake was given through the nasal tube, and no complications occurred. On the seventeenth day pharyngoscopy under local anæsthesia was performed. The feeding tube was removed and the necrotic fascia lata picked out. The tantalum gauze was firmly adherent

FIG. 274.—Radiograph of the neck, taken one year after the tracheal reconstruction.

to the underlying structures. An œsophagoscope was passed through the tantalum tube and no obstruction encountered. The feeding tube was re-inserted.

On the twenty-fourth day the feeding tube was removed. On the forty-fifth day some difficulty in swallowing had occurred and pharyngoscopy was repeated. The tantalum mesh was lying loose and was removed. The pharynx was completely epithelialized beneath this. There was no obstruction.

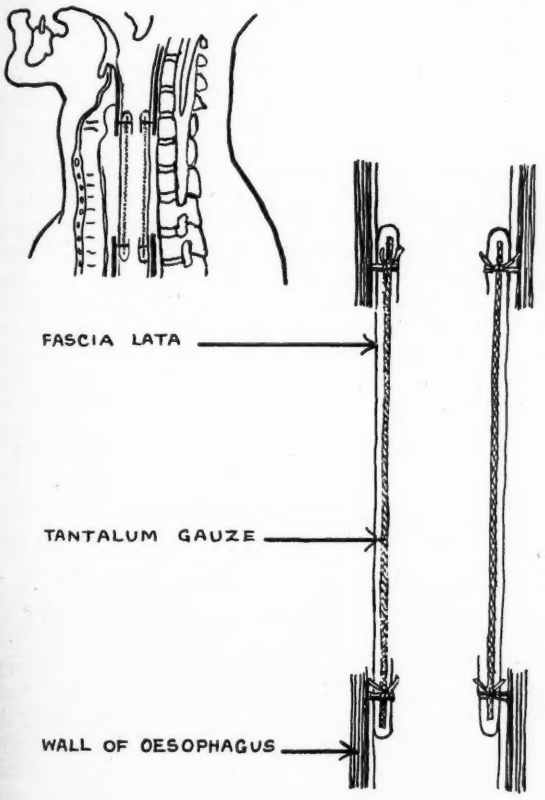

FIG. 275.—Reconstruction of the cervical œsophagus.

Three and a half months after operation the patient was well and œsophagoscopy under local anæsthesia was performed to see the state of the œsophagus.

Report: "Left recurrent nerve palsy present. The pharynx is completely epithelialized with normal-looking mucosa through which fibrous tissue can be seen. No contracture. Bougie passed into œsophagus with complete ease. The mucosa traumatizes easily, so the endoscope was not passed into the œsophagus. No evidence of recurrence."

Five months after the operation considerable difficulty in swallowing recurred. Pharyngoscopy showed a recurrence at the lower end of the repaired section of the gullet. One week later a mass in the neck was diagnosed as recurrent carcinoma. The neck was explored and the recurrence confirmed. The patient died six months after the original operation, having swallowed well for five months.

The other two patients did not live long. In both primary healing occurred, but one, a man of 74, developed bronchopneumonia as a result of the operation and died four weeks later. The second patient died five weeks after her operation, from a surgical error which provided us with a valuable lesson. During the operation both superior laryngeal nerves were divided. Her progress was satisfactory until the nasal feeding tube was removed. She could then swallow but the entrance to her larynx was without sensation so that she inhaled food. This disability ultimately proved fatal and she died from bronchopneumonia. Post-mortem examination of these two patients showed that epithelialization was nearly complete, and as the defects were 10·0 c.m. and 11·5 cm. long they provide interesting evidence of the rate at which the epithelium in this region can grow.

These four cases illustrate a method by which the trachea or cervical œsophagus can be reconstructed. The method is based on certain fundamentals. First, the epithelium of some body tubes can grow very rapidly across a large defect. Second, a temporary seal is necessary during the first few hours to limit infection while the tissue planes are being closed off. Fascia lata has proved satisfactory for this purpose. Third, a splint must be used to maintain the lumen and prevent stenosis. In the case of the trachea this should be permanent; for the pharynx and œsophagus it need only be temporary. Tantalum gauze has been used because it is a convenient material to work with, is inert in the body, and when permanently incorporated the fine mesh allows granulation tissue to grow through to its inner side where it provides a base for the growing layer of epithelium.

From our limited experience this method appears to be both satisfactory and an improvement on previous procedures (Trotter, 1913; Wookey, 1948).

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THE SURGICAL TREATMENT OF NON-RESECTABLE DUODENAL ULCER

THE ANTRAL EXCLUSION OPERATION (BANCROFT-PLENK MODIFICATION)

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The Term 'Partial Resection'.—Nowadays there is general agreement that partial resection of the stomach is the operation of choice for the surgical treatment of gastric and duodenal ulcer. This standard operation, well-planned and executed, gives the most satisfactory results; the mortality is very low and the end-results are far superior to time-consuming, yet expensive and uncertain, medical treatment. In agreement with other clinics, the material of our clinic, based on nearly 3000 resections for gastric and duodenal ulcers during 30 years, showed: 95 per cent permanently cured, with frequency of recurrent ulceration not exceeding 0·42 per cent, and with an operative mortality steadily decreasing from 6·9 per cent in 1940 to 3·1 per cent in 1947. This operation is now performed on thousands of cases, and although there is general agreement as to the indications and contra-indications, there is still much confusion as to its exact definition (which must be based on anatomical data), and this makes the comparison of statistics rather difficult.

Some surgeons, speaking of partial resection, remove one-half or two-thirds, while others remove two-thirds or three-quarters of the stomach; others speak of 'subtotal gastric resection'. Visick (1948) very rightly drew attention to the fact that it is impossible to measure accurately the size of the stomach, and that the size of the part of the stomach left would be a better criterion than the size of the removed part. He introduced the term 'measured radical gastrectomy'—and he is radical, with division of the left gastro-epiploic artery and of the greater part of the vasa brevia. On the other hand, Lewisohn (1945) calls a halt to the unjustifiable misuse of the term 'subtotal gastrectomy' in connexion with gastric resection for duodenal ulcer. The term 'subtotal gastrectomy' means, as he points out, that practically the whole organ is removed and that only a tiny portion is left behind. Such extensive gastric resections are very rare, and the common operation for duodenal ulcer should therefore never be classified as 'subtotal gastrectomy', but as 'partial gastrectomy'.

Certain criteria must, however, be observed—namely, three-quarters and more of the stomach must be removed with division of the left gastric artery near the pancreas, and division of all the branches of the left gastro-epiploic artery along the greater curvature; the remainder of the stomach must not be broader than a palm, and at the end of the operation it should retract under the ribs well away from the median incision.

This wide resection is well based on empirical and experimental data. Wangensteen and Lannin (1942) have shown clearly that if three-quarters of the stomach is removed, 63 per cent of cases become truly achlorhydric, and Heuer and Holman (1943) showed that the proportion of acidity is directly proportional to the amount of stomach removed.

Visick (1948) showed clearly that the incidence of recurrent ulcer was in direct relation to the extent of the gastrectomy and the blood-supply of the gastric remnant, being 5·3 per cent in cases where only half of the stomach was removed (Heuer, 1944), 3·7 per cent where the left gastro-epiploic artery was partly divided, and nil in the cases where all branches of the left gastro-epiploic artery with some branches of the vasa brevia were divided.

THE NON-RESECTABLE DUODENAL ULCER

While this standard operation of partial resection is applicable to all cases of resectable duodenal ulcers, there exist quite a number of non-resectable ones, where the surgeon faces a dilemma as to the choice of the proper operation, a question which is still the subject of lively discussions. The frequency of such non-resectable ulcers is considerable and depends among others on nutritional and geographical conditions, etc. Their importance lies in the fact that they produce more intensive subjective symptoms with greater hyperacidity, they have more tendency to bleed, and they are most resistant to medical treatment.

Table I.—Variations in Percentage Occurrence of Non-resectable Ulcers*

Author	*Percentage*
Schewket	65
Fromme	50
Kirschner-Philippides	50
Jacobovici	50
Denk	32
Geroulanos-Tool	21
Schubert	20
Zukschwerdt and Eck	19
Smparounis	12
Demel	10
Hoche and Marangos	8·5
Berger	8
Alevisatos	7·6

* Figures as given by Zukschwerdt and Horstmann, *Ergebn. Chir. Orthop.* 1936, **29**, 440 (supplemented).

As the surgeon's experience increases, the number of non-resectable duodenal ulcers decreases—for example: Finsterer from 16·6 to 6 per cent; Florken from 20 to 10 per cent; Haberer from 16·8 to 6 per cent; while Nissen, among 220 operations for duodenal ulcers (1938), reports only 3 cases in which the term 'non-resectable' was justifiable.

Apart, however, from some general conditions, such as adiposity, cardiovascular disorders, etc., some anatomical criteria must support the term 'non-resectable duodenal ulcer': (*a*) Involvement

of ampulla of Vater or its vicinity in the ulcerated area; (*b*) Extensive penetration into the pancreas or hepatoduodenal ligament, with probable involvement of the common duct; (*c*) Perforation into the gall-bladder.

The late war showed that the incidence of ulcer generally, and especially that of non-resectable duodenal ulcers, considerably increased (England, Tidy, 1943; Germany, Schubert, 1947; Holland, Borema, 1948). In Greece the percentage of non-resectable duodenal ulcers during the starvation period 1941–3 increased at our clinic from 20 to 30 per cent and fell back again after 1944 to 20 per cent.

Paraskevas found in the Piræus material during the same period an increase up to 40 per cent and in Cyprus the percentage was as high as 32 per cent. Comparing the ulcer material in other countries, such as England, one is impressed that in our material ulcers are much more common, more severe, with a higher rate of penetration. This is due to the fact that most people are undernourished and come to operation after many years of suffering, and the co-existing gastritis is always very severe (Kourias, 1946).

In all these non-resectable cases we were obliged until some years ago either to attempt the resection of the ulcer-bearing part of the duodenum, with a high rate of mortality, due either to uncertain duodenal occlusion or damage to bile or pancreatic ducts, or to be satisfied with a simple gastro-enterostomy, well aware of the minor benefits and major disadvantages of this procedure. This ' bad operation ', which gave in our clinic a mortality of 9·3 per cent and 15 per cent recurrent ulcerations (anterior anastomoses 13·6 per cent, posterior anastomoses 4·6 per cent), is now almost totally abandoned, being reserved for only those very rare and exceptional cases where the large ulcer causes such a wide infiltration of the prepyloric antrum that every other form of operation is practically impossible.

We have never used a two-stage procedure in which the first stage is a high gastrectomy with the old type pyloric exclusion, followed after an interval of a few weeks by resection of the pylorus and duodenum, although a successful series of this type has recently been reported by McKittrick, Moore, and Warren (1944).

For all other non-resectable cases we use the exclusion operation introduced by Finsterer in 1918, and since 1928 this has replaced gastro-enterostomy. From our experience of this method, we can now say that this operation is as beneficial as the radical partial resection, and that the final results collected and published from our clinic (Paraskevas and Sapkas, 1948) are not inferior to those achieved by the partial resection.

The Finsterer exclusion operation, based on experimental studies, was for years the centre of lively controversy, and the cause of disagreement lay in the variable importance attributed to the pyloric canal, its muscle, and its relation to the development of post-operative recurrent ulceration. The operation leaves behind part of the pars pylorica, from which the acid-stimulating hormone ' gastrin ' (Edkins) comes. The very long controversy, headed chiefly by Haberer (1938), the high rate of recurrent ulcers (Haberer, 1938; Ogilvie, 1938; Keefer, 1942; and others), and the complications (bleeding, perforation, leakage) caused us for many years to be rather critical towards this operation.

It is known that the Finsterer exclusion operation can be done in two ways: either by removal of the pylorus, or, in cases where the ulcer lies very near to the pylorus, by resection about two finger-breadths above it. This second method is preferable and Leriche (1938) believes that the advantage of this method lies in the satisfactory suture of the remnant of antral pouch. Sénèque (1936) advises division of the stomach 5–6 cm. from the pylorus, in order to obtain easier invagination, while Ameline (1945) resects a part of the pyloric sphincter for the same purpose. We also had in the beginning our difficulties when we were performing this exclusion operation with conservation of the pylorus after the classic method of Finsterer. Duodenal leakage occurred on several occasions, due either to blowing up, through increased pressure of mucus secretion, or to blood accumulation in the blind prepyloric pouch, or to unsafe suture. The inversion was rather difficult, sometimes even impossible, because the transverse diameter of the stomach diminishes in the direction of the duodenum. If the inversion was forced, sometimes the stump became anæmic, and we were compelled to proceed to the unpleasant and most difficult radical removal of the ulcer to avoid leakage. Bancroft's (1932) suggestion that the antral mucosa should be cored out if the pylorus is left, was an attempt to overcome difficulties, and was gladly accepted.

All these troubles ceased to exist for us when we used for the closure of the prepyloric pouch the method described by Plenk in 1936, which is simple, sure, and, it seems, very little known in Anglo-Saxon countries.

As time went by and experience was gained, we became more enthusiastic about this operation, and since 1937 it has been the operation of choice for the non-resectable duodenal ulcer in our clinic.

Certain precautions are, however, necessary for the success of the operation. Makkas showed that it is possible in 80 per cent of cases by careful X-ray examination to say if the ulcer is resectable or not. If, after opening the peritoneal cavity, inspection proves that the ulcer is non-resectable then the antral exclusion operation must be carried out. It is a bad mistake to try to mobilize the duodenum, cut the blood-supply, and then, seeing the impossibility of radical resection, conclude the operation as a resection for exclusion, for necrosis, leakage, and bleeding are likely to occur.

Further, the vessels of the prepyloric part must not be ligated. In the coning-out of mucosa, bleeding is not very troublesome; however, careful hæmostasis is essential. In the post-operative period the danger of blowing up of the duodenal stump through gastric distension from stasis and blood accumulation must be avoided through the regular use of Ryle's tube.

Technique.—The vessels of the greater and lesser curves are ligated, starting 2 in. proximally to the pylorus. Circular division between clamps is made of the seromuscular coats of the antrum,

3–4 cm. above the pylorus (two fingerbreadths). The mucosal cone is separated down to the pylorus, and ligation of the base of the cone is made. It is easier to divide the stomach between clamps two fingerbreadths above the pylorus (*Fig.* 276). The divided stomach is covered and reflected to the left.

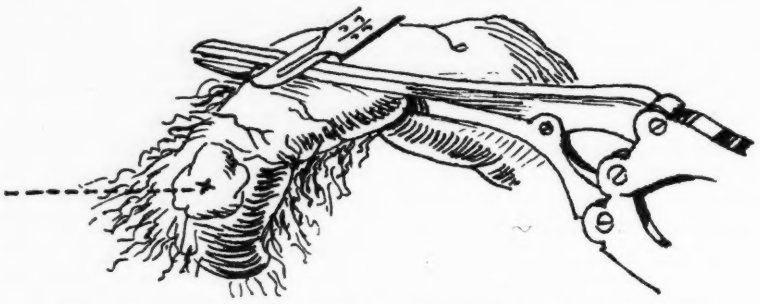


FIG. 276.—Division of the stomach two fingerbreadths above the pylorus.

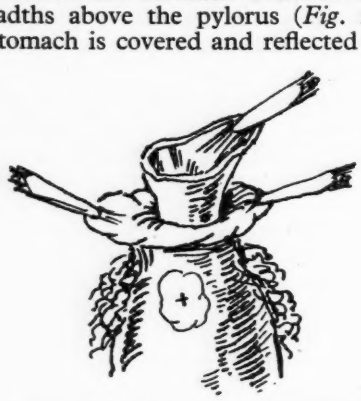
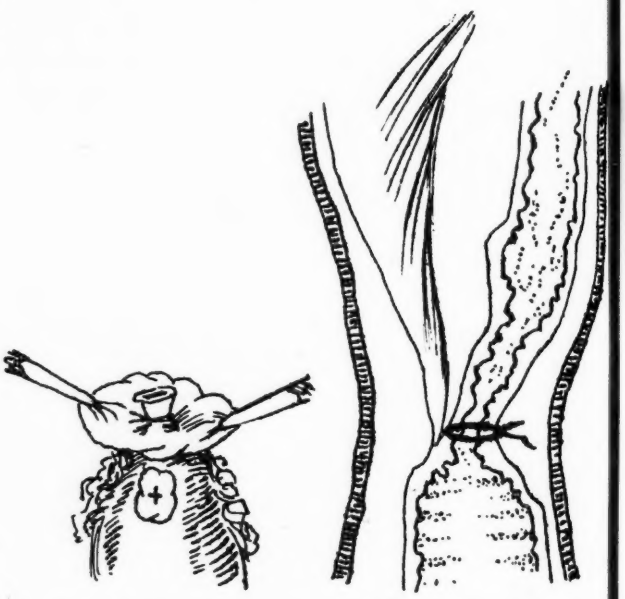


FIG. 277.—The mucous membrane of the prepyloric antrum is grasped with Allis forceps and, partly by blunt and partly by sharp dissection, is cored out from within as far as the pylorus itself.

The mucous membrane of the prepyloric antrum is grasped with Allis forceps and, partly by blunt, partly by sharp, dissection, is cored out from within, as far as the pylorus itself (*Fig.* 277). The cone of mucosa is transfixed at the base as deep as possible, the rest of the mucosa being removed in the manner suggested by Bancroft (*Figs.* 278, 279).



FIGS. 278, 279.—The cone of mucosa is ligated and transfixed at the base, the rest of the mucosa being removed.

FIG. 280.—Closure of the anterior seromuscular cuff of prepyloric antrum to the posterior by means of three rows of sutures.

Plenk's method concerns the manner of closing the small pyloro-antral pouch. This closure is done from within, without invagination, by means of three rows of sutures—the first quite close to the pylorus unites with fine catgut stitches the posterior seromuscular duodenal wall to the anterior (personally we prefer a purse-string); the second row unites with interrupted stitches the anterior to the posterior wall, obliterating the cavity which remains; and the third row with silk unites the cut end of the antrum (*Fig.* 280). Careful hæmostasis is absolutely essential and the stump is covered with omentum.

Discussion.—This Plenk technique described in 1936 is little known. Wangensteen (1942) describes a similar technique by which leakage can be avoided. Similar techniques with slight modifications were described by Allen (1942), Kleinschmidt (1946), Schubert (1947), Wolfson and Rothenberg (1938), Allen and Welch (1942), Ogilvie (1947), while Carayannopoulos has used a similar technique since 1932.

If the antral exclusion operation by the Bancroft-Plenk method is typically and elegantly executed, with conservation of the pylorus, we believe that this is the operation of choice for the non-resectable

duodenal ulcer, in contrast to the exclusion operation with removal of the pylorus, in which it is certain that the closure of the duodenum proximal to an ulcer that remains untouched can seldom be executed satisfactorily. Very often the separation of the duodenum takes place very close to the ulcer, between infiltrated and œdematous tissue, and inversion is either difficult or impossible, and, as we see in the statistics, the sutures are not reliable.

If an operation is of daily routine use, certain conditions must be fulfilled :—

1. It must not be technically very difficult, and certainly not the privilege of certain surgeons only.
2. The dangers of operation must not be disproportionally heavier in comparison with the gravity of the disease.
3. The operation must give the best possible long-term results.

Statistics.—On account of the rich ulcer material in our clinic, we think we are able to prove that the antral exclusion operation with conservation of the pylorus fulfills all the above conditions, as shown in the following statistics of our clinic.

Table II.—TOTAL NUMBER OF GASTRIC AND DUODENAL ULCERS OPERATED UPON FROM JANUARY, 1931, TO MAY 31, 1947, AT THE SURGICAL CLINIC, RED CROSS HOSPITAL, ATHENS

	Gastric Ulcers	Duodenal Ulcers	Marginal Ulcers	Total
First period, 1931-1941 :—				
1931	6	54	5	65
1932	8	67	5	80
1933	11	94	14	119
1934	10	77	12	99
1935	10	76	9	95
1936	5	65	2	72
1937	10	75	6	91
1938–9	10	115	7	132
1939–40	11	88	7	106
1940–1	4	84	2	90
1931–41	85	795	69	949
Second period, 1941–1947 :—				
1941–2	18	105	2	125
1942–3	24	201	6	231
1943–4	13	318	6	337
1944–5	15	163	5	185
1945–6	20	324	8	352
1946–7	18	264	18	300
1941–47	108	1375	45	1530
1931–47	193	2170	114	2479

Table II shows clearly the great frequency of ulcer operations in our clinic, especially since the occupation of Greece in 1941. Needless to say, we are very rigorous regarding the indications for operation, and most cases are sent to us after several attempts to cure the ulcer medically have failed.

The second point of interest is the relation of gastric to duodenal ulcer, being, in our clinic, 1 : 11.

Table III shows that in recent years gastro-enterostomy has been seldom performed in the clinic, and its place has been taken by the exclusion operation. The percentage of exclusion operations in relation to operated duodenal ulcers was 10·6 per cent in the first period, increasing to 20·6 per cent during the second period 1941–7. At the same time, the radical partial resection was employed in 67·4 per cent in the cases operated up to 1941, increasing to 77·3 per cent between 1941 and 1947. In the year 1946–7, 89·6 per cent of duodenal ulcers were radically resected. Generally, in 2170 cases of duodenal ulcers, the following procedures were employed :—

1599 cases underwent	partial resection	73·6	per cent
369 „ „	exclusion operation	17·0	„ „
202 „ „	gastro-enterostomy	9·3	„ „

There are surgeons who say that all duodenal ulcers can be resected, and therefore there is no need for palliative operations. This statement is not true. There are certainly a number of non-resectable duodenal ulcers, but, in addition, there are ulcers the resection of which is either very difficult or dangerous. The surgeon who happens to injure the bile or pancreatic duct in the resection of a duodenal ulcer situated well to the right will be more hesitating in another similar case, and he will prefer not to perform a radical resection again, if he has at his disposal an operation that is sure, simple, almost without danger, and which gives excellent results. Therefore, we believe that the antral exclusion operation by the Bancroft-Plenk modification is a very satisfactory operation, giving very good results. If we prefer this operation in the difficult cases, this is done not merely to avoid technical difficulties, but rather in the interests of the patient whom we have to protect from unnecessary dangers.

Table III.—SHOWING THE TYPE OF OPERATION FOR DUODENAL ULCER

Total of Operation Cases	Wide Resection	Exclusion Operation		Gastro-enterostomy
		With Removal of Pylorus	With Conservation of Pylorus	
First Period, 1931–41 :—				
795	536 (67·4 per cent)	29	56, of which 31 cases had the Plenk operation	174 (21·8 per cent)
		10·6 per cent		
Second Period, 1941–7 :—				
1375	1063 (77·3 per cent)	71	213, of which 212 cases had the Plenk operation	28 (2·1 per cent)
		20·6 per cent		
2170	1599 (73·6 per cent)	100	269, of which 243 cases had the Plenk operation	202 (9·3 per cent)
		17 per cent		

The mortality of all cases operated upon for duodenal ulcer has fallen from 6·9 per cent during the first period (1931–41) to 3·5 per cent during the second period (1941–7). The greatest mortality was found with gastro-enterostomy because this operation was executed only in the very bad and risky cases.

The radical resection showed a decline in mortality from 6·9 per cent during 1931–41 to 3·1 per cent during 1941–7.

The most interesting point of this table is the mortality-rate for the antral exclusion operation. Of 269 antral exclusion operations with conservation of the pylorus, 26 were performed after Finsterer's original classical method with invagination of the stump and there was a mortality of 15 per cent.

Table IV.—MORTALITY OF DIFFERENT OPERATIONS IN THE TREATMENT OF DUODENAL ULCERS

TYPE OF OPERATION	NO. OF CASES	MORTALITY	PER-CENTAGE
First Period, 1931–41 :—			
Radical resection	536	37	6·9
Antral exclusion operation with conservation of pylorus. Classic method of Finsterer	25	3	12·0
Antral exclusion operation with conservation of pylorus. Bancroft-Plenk modification	31	0	—
Exclusion operation with removal of pylorus	29	1	3·4
Gastro-enterostomy	174	14	8·0
	795	55	6·9
Second Period, 1941–7 :—			
Radical resection	1063	33	3·1
Antral exclusion operation with conservation of pylorus. Finsterer's classic method	1	1	100·0
Antral exclusion operation with conservation of pylorus. Bancroft-Plenk modification	212	5	2·3
Exclusion operation with removal of pylorus	71	6	8·4
Gastro-enterostomy	28	4	14·2
	1375	49	3·5

All the other 243 cases were operated upon according to the modification of Bancroft-Plenk with a mortality of 2·3 per cent, and the difference between the original classical operation of Finsterer and that of the modification of Bancroft-Plenk is truly very remarkable. Further, we notice that the exclusion operation with removal of the pylorus, for the above-stated reasons, is much more dangerous, with a mortality of 7 per cent. Other mortality statistics for the Bancroft-Plenk operation on a great number of cases do not exist, except those of Plenk, who reported on 116 cases with 3 deaths (2·6 per cent).

Table V.—RESULTS OF THE BANCROFT-PLENK OPERATION

	NO. OF CASES	MORTALITY	PERCENTAGE
Makkas Clinic	243	5	2·3
Marangos (Limassol Hospital)	30	0	—
Paraskevas (Pireaus Clinic)	26	1	2·6
Plenk (Linz Hospital)	116	3	—
TOTAL	415	9	2·1

In *Table V* we have added these cases to the cases of Paraskevas and Marangos.

Paraskevas and Sapkas from our clinic examined and published (1948) the cases of duodenal ulcer where the exclusion operation was performed. *Tables VI, VII* show the results of their examination, which are most interesting and encouraging.

These statistics show that 95 per cent of the cases were definitely cured after the antral exclusion operation without removal of the pylorus, by the Bancroft-Plenk method. All these cases were very pleased with the results of operation; some put on weight up to 25 kg. Some of them were even working harder than before and stood the starvation period well. *No suspicion of recurrent ulceration was noted*, although some of them had been operated upon several years ago, and although we have operated on quite a large number of jejunal peptic ulcers after gastro-enterostomy, and even some rare cases after radical resection (Makkas and Marangos, 1936).

Table VI.—NUMBER OF CASES OPERATED ON AND RE-EXAMINED

Number of cases operated on	369
Post-operative deaths	16
Remaining cases	353
Addresses of 18 lost, so letters sent to	335
Re-examination or answer received from	221 (65·9 per cent)

Regarding our two unsatisfactory cases :—

The first case, three weeks after operation, had severe bleeding. In an urgent operation we removed the duodenal stump with the ulcer; the patient was cured, and has been perfectly well since then.

The second case, three months after operation, had attacks of cholangitis with rigors. Operation 18 months after the first operation showed that the duodenal stump and the hepatoduodenal ligament were fibrosed and the bile-duct distended—probably due to the cicatrization. After a choledochoduodenostomy, the patient was permanently cured.

Table VII.—END-RESULTS OF RE-EXAMINATION OF 221 CASES OPERATED UPON FOR DUODENAL ULCER IN WHOM THE EXCLUSION OPERATION WAS PERFORMED

	With Conservation of the Pylorus (177 cases, of which 158 operations were Bancroft-Plenk)		*Removal of Pylorus* (44 cases)	
Excellent results	126 (71·3 per cent)	95 per cent	28 (63·6 per cent)	
Good results	42 (23·7 „ „)		15 (34·0 „ „)	
Satisfactory	7 (3·9 „ „)		1 (2·2 „ „)	
Bad	2 (1·1 „ „)		—	
	177		44	

One may object that not all cases were re-examined, but we must remember that this is a difficult task after the war—especially in Greece, after years of occupation, with a still-raging civil war, with such a wide movement of population and interruption of communications—and therefore the percentage of 65·9 re-examinations must be considered satisfactory. There is no reason to believe that the results in the non-examined patients are different from those who underwent an examination, especially as here the operated cases commonly present themselves for re-examination by the surgeon who performed the operation.

The re-examination of the 30 Limassol cases operated during 1940–7 was easier and complete. All the 30 cases were followed up systematically and were personally re-examined (G. M.).

The *results* are as follows :—

Excellent	25
Good	3
Satisfactory	1
Bad	1
TOTAL	30

One patient complained of dumping symptoms, which are improving. The other case had, three months after the Bancroft-Plenk operation, moderate melæna. Since that time he has had no complaints.

There are very few statistics of end-results after antral exclusion operations. Wangensteen (1942) considers the antral exclusion operation a satisfactory and effective operation for the irremovable or inoperable duodenal ulcer, and the reason it has not come into general favour among gastric surgeons is because of the likelihood of leakage from the residual antral pocket.

Schubert (1947) has 47 cases without mortality, of which only 2 have had slight trouble.

Ramb (1948) praises the technique of Plenk and remarks that the distant results of exclusion operations are nearly similar to those of partial resection.

Lannin (1945) finds the antral exclusion operation with excision of mucosa a satisfactory surgical process.

Kleinschmidt (1946), Allen and Welch (1946), McKittrick (1944), Rienhoff (1945), and Vasconcelos (1947) are using the antral exclusion operation, and the incidence of recurrence of ulcer following antral exclusion is less than that in the group of resections as a whole. Wangesteen reports no stomal ulcers following antral exclusion in a series of just under 100 cases.

Watson (1947), in re-examination, found in 30 cases after pyloric exclusion excellent results in 46·7 per cent, good in 36·7 per cent, and improved in 10 per cent. Although in his cases the test-meals showed the presence of free acid and a much higher quantity of both free and total acid when compared with those test-meals showing free acid after routine gastrectomy, no recurrent ulcerations were noted.

Maingot (1948) quotes only 2 per cent of recurrences after this operation, which he recommends as a simple, safe, and excellent procedure for the large, fixed, penetrating, irremovable ulcer of the posterior wall of the duodenum.

Finsterer (1940) quotes a similar figure, but without reference to acidity.

Gardner and Hart (1948), at the meeting of the Southern Surgical Association, December, 1947, in a comparative study of the results of gastro-enterostomy, gastric resection, and vagotomy, found that the operative procedure may be difficult when an active ulcer with posterior penetration has attached itself to the pancreas or portal triad. Five of the 8 deaths in selective resections were caused by leakage at the duodenal stump or damage to adjacent structures. This experience has influenced their decision to exclude 35 per cent of duodenal ulcers when resection is done. At follow-up, only 1 in the group of 23 antral exclusions had a recurrent ulcer. The mucous membrane, however, in this case had been left in the pyloric stump. Hence their present practice of removing antral mucosa in all cases.

In the discussion Grove (1948) stated that he used the Bancroft modification of the Finsterer exclusion method in the cases where removal of the ulcer has not been feasible. Bancroft (1948), who modified the Devine operation for pyloric exclusion with excision of the mucosa of the antrum, reported that throughout the years he has operated upon quite a number of patients with excellent results, with the exception of one case which bled post-operatively and had to have the duodenal stump resected. Where the ulcer is low in the duodenum and one is afraid of the possibility of duodenal leakage, this operation is well worth considering. Since his report in 1932 he has modified the procedure by resecting the medial portion of the stomach, leaving enough antrum so that the mucosa can be cored out and the stump closed without tension.

Hart, closing the discussion, said that the greatest operative risk lies in resection of a duodenal ulcer densely adherent to or ulcerated into the pancreas, particularly when located some distance below the pylorus. In his series, the results following exclusion operations have been as good as following resections. Since such an operation with removal of the antral mucosa avoids all the special risks associated with resections of perforated or adherent duodenal ulcers in proximity to the bile-duct, it probably should be used more often. This is particularly true for those operators who do some stomach surgery but who are less experienced in handling these complicated and difficult situations, and he was favourably impressed by the percentage of excellent results following resections or exclusion types of operations.

At several meetings of the Greek Surgical Society, February to April, 1948, as a result of the report by Kourias on the surgical treatment of ulcer it was discovered that many Greek surgeons (Alevisatos, 1948; Carayannopoulos, Kourias, Koufas, Manos, Palios, Paraskevas, Sapkas, Sparounis, Tool, and others) were using the Bancroft-Plenk modification in the treatment of non-resectable duodenal ulcer and that they found the procedure simple, safe, and excellent, yielding most gratifying late results.

In conclusion, we want to draw attention to the fact that the antral exclusion operation by the Bancroft-Plenk modification, with a mortality of 2·3 per cent and with end-results satisfactory up to 95 per cent, is a very satisfactory operation which, when well executed, gives in cases of non-resectable duodenal ulcers, with the least risk, final results much superior to any other procedure and nearly approaching those of partial resection.

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THE PROBLEM OF THE LOCALIZED SURFACE INTRACRANIAL HÆMORRHAGE CAUSING COMPRESSION OF THE BRAIN*

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THIS paper is concerned with the study of 18 consecutive cases of compression of the brain by localized massive surface hæmorrhages following trauma to the head. All the cases were treated personally in the Newcastle upon Tyne Neurosurgical Unit. Fifteen cases were examples of massive extradural blood-clots and 3 were examples of localized massive subdural clots. In this series of 18 cases there were 3 deaths—a recovery rate of 83 per cent. It is proposed to begin with a consideration of the extradural hæmorrhages.

EXTRADURAL HÆMORRHAGES

The fifteen cases of extradural hæmorrhage were seen and operated upon over a period of five years. The actual incidence of this type of hæmorrhage in any large series of head injuries is small—approximately 1–2 per cent—but it is an extremely important group of cases, for with timely diagnosis and treatment the results obtained in treating this type of hæmorrhage are among the most satisfactory in surgery—for not only is a life saved but the patient recovers completely and thus morbidity rates are minimal. In this personal series of 15 extradural hæmorrhages there were 3 deaths—a recovery rate of 80 per cent.

In its classical form an extradural hæmorrhage is characterized by a latent period which intervenes between the initial trauma and the onset of symptoms of cerebral compression. However, cases are encountered in which a true latent period is absent. In such cases a blow to the head results in concussion, and thereafter the clinical state is one of steadily deepening unconsciousness. This type of clot is the one most likely to be missed unless careful routine observation of the unconscious patient is undertaken. In this series of extradural hæmorrhages 10 cases presented a characteristic latent period, while 5 cases had no latent period.

CASES PRESENTING A CHARACTERISTIC LATENT PERIOD

(*Table I*)

In this group there were 8 males and 2 females and their ages varied from 7 to 45 years.

The period of initial unconsciousness following trauma to the head was absent in 4 of the 10 cases and short in the remaining 6 cases—varying from a dazed sensation of only a few seconds' duration to actual unconsciousness for a few minutes. This initial unconsciousness following trauma to the head is due to a mild, diffuse, reversible, neuronal injury.

The latent period varied from half an hour to four days. In 5 of the cases the latent period was six hours or less. In 4 cases the latent period was twenty-four hours, and in 1 case the latent period was four days. The latent period depends upon the size of the bleeding vessel and also on the nature of the bleeding—whether arterial or venous.

The first symptom of cerebral compression. Eight of the 10 cases complained of severe headache as their first symptom. One patient became mentally confused simultaneously with the onset of severe headache and in the remaining case the first symptom of cerebral compression is not known. This headache is a most striking phenomenon if a patient suffering from it is seen before the onset of deep drowsiness. It is extremely painful, the patient becoming very irritable and resentful of any interference. He lies clutching his head, rolling from side to side, and refuses absolutely to co-operate in any examination. This phase may be short,

* Based upon a paper read to the Society of British Neurological Surgeons in Newcastle upon Tyne, Nov. 26, 1948.

however, being replaced by deep drowsiness going on to unconsciousness.

Epilepsy. Only one patient (*Case* 9) in this series suffered from epilepsy—a single generalized fit. There were no instances of Jacksonian epilepsy.

Physical Signs.—As the clot increases in size drowsiness gives way to unconsciousness, and associated with this there appear signs of pyramidal involvement on the opposite side of the body. Thus, in a typical case, the observer will note weakness and spasticity of the opposite limbs, with associated signs of pyramidal tract involvement. This developing hemiparesis may or may not be associated with convulsions—general or focal. Respiration at first is fast and deep—later becoming slower and then irregular. The pulse-rate may or may not be slow in the fully established case. Fixation and dilatation of the pupil on the same side as the clot may or may not be present, but when present it is usually a late sign and is an indication for prompt intervention.

When the above picture is seen or is known to have occurred, lateralization of the clot is a simple matter, but it so often happens that patients are brought into hospital who have progressed beyond this stage and present bilateral signs of pyramidal involvement, with or without fixation and dilatation of one or both pupils, so that it may well be impossible to lateralize the clot on physical neurological signs alone, unless the sequence of events is known. This occurs particularly when the latent period is short (a few hours or less) and when a large vessel such as the trunk of the middle meningeal artery is ruptured. In such cases the clot increases in size rapidly and the various stages described above merge rapidly into each other so that any particular phase is fleeting and easily missed. In the present series it will be seen that rapid deepening of unconsciousness associated with early development of bilateral pyramidal signs was most frequently seen in examples of extradural clots situated in the temporobasal and occipito-cerebellar regions of the skull, and this is due to early and rapid brain-stem compression.

On the other hand, in cases in which the latent period is long or in which deep drowsiness gives way to deepening unconsciousness after several days, there may be few or no localizing neurological signs at all, owing to the ability of the brain to adapt itself to slowly developing cerebral compression. This is perhaps best exemplified in the chronic subdural hæmatomas, where localizing neurological signs are the exception rather than the rule.

In this series the typical picture was seldom seen. Thus, in these 10 cases which were typical with regard to having a well-defined latent period, only 3 cases (*Cases* 5, 8, 10) showed signs of weakness on the opposite side of the body. *Case* 5 had a flaccid hemiplegia on the right side; *Case* 8 had weakness of the right arm and hand; *Case* 10 had a left hemiparesis. These three cases, in addition, had fixed dilated pupils on the same side as their clots. *Case* 6 presented no localizing neurological signs and no abnormality of the pupils. In this case the latent period was four days. Radiographs of the skull did not reveal a fracture and there was no superficial scalp bruising to aid in lateralizing the clot. At operation, after a negative exploration on the right side, a huge extradural clot was exposed overlying the left temporal lobe on the convexity, and at this exploration a very fine oblique fracture could be seen crossing the squamous temporal bone. On clinical grounds there could be no doubt that this patient had a clot, although there were no localizing signs at all. The remaining 6 cases presented bilateral spasticity of equal degree—2 patients indeed were in the state of decerebrate rigidity on admission.

Of the 2 cases which were decerebrate on admission, *Case* 4, a child aged 7 years, had a right fixed dilated pupil. She made a complete recovery, after a stormy convalescence, when her clot had been removed. The other case, *Case* 7, a young adult, had both pupils fixed and dilated, and this clot was localized by the presence of bruising in the left temporal region. A huge clot was removed at operation, but the patient died forty-eight hours later.

Of the remaining 4 cases which showed bilateral spasticity, only 1 had a unilateral fixed dilated pupil (*Case* 1) on the same side as the clot. Another case (*Case* 9) had both pupils fixed and dilated and this clot was localized by the presence of bruising in the left temporal region. This patient died forty-eight hours after removal of the clot. The remaining 2 cases presented no pupillary abnormalities and the clots in these cases were localized by evidence of local bruising on the scalp (*Cases* 2, 3). In those cases which had a unilateral fixed dilated pupil—5 of the 10 cases—clots were always found on the side of the fixed pupil.

Signs of Scalp Bruising or Hæmatoma Formation indicative of Underlying Skull Fracture.—The presence of external scalp bruising, œdema, or hæmatoma formation proved to be an excellent lateralizing sign in these cases. It occurred in 8 of the 10 cases. In one case (*Case* 8) there was no sign of external injury to the scalp, but this was a typical case otherwise. In *Case* 6, also, there was no external evidence of injury—this case has been referred to earlier—there were no localizing signs, and the clot, a very large one, was located by repeated burr-hole explorations.

Radiography of the Skull.—Radiographs of the skull, if available, may be helpful in diagnosis, particularly for lateralizing purposes. Yet at times there is no demonstrable fracture at X-ray or at operation and still there is an extradural clot present. This occurred on two occasions (*Cases* 5, 9). Both cases were clear-cut examples of extradural clot compression and at operation large clots were removed. In *Case* 5 there was no fracture to be seen at operation or on the radiographs, and in *Case* 9 no fracture could be demonstrated on the radiographs, at operation, or at post-mortem examination. The diagnosis of extradural hæmorrhage is essentially a clinical diagnosis—if radiographs cannot be produced rapidly they are valueless. Delay, for any reason, jeopardizes the patient's life.

Source of Bleeding.—The source of bleeding was identified in all ten cases. In 6 cases the bleeding vessel was the trunk of the middle meningeal artery. In 2 cases the venous channels which accompany the middle meningeal artery were

Table I.—Massive Extradural Hæmorrhages with Latent Period

Case No.	Age	Sex	Period of Initial Unconsciousness	Latent Period	First Symptom of Cerebral Compression	External Scalp Bruising	Fracture of Skull	Pupils	Neurological Signs	Pulse	Temperature °F.	Respiration	Findings at Operation		Result
													Site of Hæmorrhage	*Source of Hæmorrhage*	
1	43	M.	Dazed for few secs.	2 hr.	Severe headache	Bruising, œdema, left temporal region	Left parietal	Left, fixed and dilated	Bilateral spasticity	54	101	20	Left temporo-basal	Artery (trunk of M.M.A.)	Recovery
2	11	M.	Dazed for few secs.	24 hr.	Severe headache	Bruising, right temporal region	Right squamous temporal	Normal reactions	Bilateral spasticity	100	97	24	Right temporo-parietal	Vein (M.M.V.)	Recovery
3	22	M.	Few mins.	24 hr.	Severe headache	Bruising, œdema, left mastoid region	'Springing' of left occipito-parietal suture	Normal reactions	Extreme bilateral spasticity	140	97	28	Left occipito-cerebellar	Large diploic channel	Death
4	7	F.	—	6 hr.	Not known	Hæmatoma, right parietal region	Right squamous temporal	Right, fixed and dilated	Decerebrate rigidity	100	97	Cheyne-Stokes	Right temporo-basal	Artery (trunk of M.M.A.)	Recovery
5	28	M.	—	½ hr.	Severe headache and mental confusion	Bruising, œdema, left temporal region	No demonstrable fracture	Left, fixed and dilated	Flaccid paralysis right side	48	98	20	Left temporo-basal	Artery (trunk of M.M.A.)	Recovery
6	27	M.	Dazed for few secs.	4 d.	Severe headache	—	Left squamous temporal (very fine crack)	Normal reactions	Nil	48	98	24	Left temporal (convexity)	Artery (posterior branch of M.M.A.)	Recovery
7	25	M.	—	3 hr.	Severe headache	Bruising, œdema, left temporal region	Left parieto-temporal	Both, fixed and dilated	Decerebrate rigidity	78	97	Cheyne-Stokes	Left temporo-basal	Artery (trunk of M.M.A.)	Death
8	15	M.	Dazed for few secs.	24 hr.	Severe headache	—	Left temporal	Left, fixed and dilated	Paresis right arm and hand	50	98	24	Left temporo-basal	Artery (trunk of M.M.A.)	Recovery
9	25	F.	—	3 hr.	Severe headache	Bruising, œdema, left temporal region	No demonstrable fracture	Both, fixed and dilated	Extreme bilateral spasticity	96	101	Cheyne-Stokes	Left temporo-basal	Artery (trunk of M.M.A.)	Death
10	45	M.	Dazed for few secs.	24 hr.	Severe headache	Bruising, œdema, right temporal region	Right squamous temporal	Right, fixed and dilated	Left hemiparesis	48	97	20	Right temporal (convexity)	Vein (M.M.V.)	Recovery

ruptured. In 1 case a branch, the posterior branch, of the middle meningeal artery was ruptured. In the remaining case the bleeding came from a large diploic channel.

In those 6 cases in which the trunk of the middle meningeal artery was found to be bleeding, the latent period was six hours or less in 5 cases. In the remaining case the latent period was twenty-four hours. All these six clots were situated in the temporobasal region of the skull (i.e., in the middle fossa), and each case developed early dilatation and fixation of one or both pupils—2 patients in fact died. A short latent period is thus strong presumptive evidence of bleeding from the trunk of the middle meningeal artery, and is a strong indication for very prompt action. The longer latent period in the sixth case (*Case* 8) was, no doubt, due to temporary sealing of the vessel resulting in arrest of hæmorrhage—with recurrent hæmorrhage later due to recovery from shock or to straining on the part of the patient. These temporobasal extravasations in the middle fossa appear to be extremely dangerous injuries because they result in early brain-stem compression and death if not relieved quickly. In this series the same appears to apply to clots in the occipito-cerebellar region, e.g., *Case* 14 (to be described later), who, eight hours after a blow on the head, was in a state of decerebrate rigidity with Cheyne-Stokes respiration. Following removal of the clot he remained decerebrate for two days before beginning to recover. *Case* 3, a fatal case, and another occipito-cerebellar hæmorrhage, stopped breathing for forty minutes before operation was possible and had to be kept going with artificial respiration.

With hæmorrhages over the convexity of the brain, 2 cases had latent periods of twenty-four hours (*Cases* 2, 10) and 1 case (*Case* 6) had a latent period of four days. All had massive hæmorrhages but from vessels of small calibre, and so, presumably, bleeding had proceeded at a slow rate and the development of cerebral compression had been more gradual than in the cases mentioned above in which large clots had formed rapidly from the middle meningeal trunk.

CASES IN WHICH THERE WAS NO LATENT PERIOD

(*Table II*)

There were 5 examples of extradural hæmorrhage in this group. In this type of case there has usually been a somewhat more severe intrinsic injury to the brain and some cases may be complicated by subdural and subarachnoid bleeding. Diagnosis in this type of case may be difficult. All these five cases were males and their ages varied from 13 to 55 years.

With the exception of *Case* 14, who, following a blow on the head, remained unconscious until operated upon eight hours later, these cases had remained unconscious for a period of days. *Case* 14 is included in this group simply because there was no latent period; but, apart from this, it was an instance of a rapidly advancing cerebral compression resulting in a condition of decerebrate rigidity with a right, fixed, dilated pupil. This clot was situated in the occipito-cerebellar region and

Table II.—Massive Extradural Hæmorrhages without Latent Period

Case No.	Age	Sex	Period of Initial Unconsciousness	Latent Period	First Signs of Cerebral Compression	External Scalp Bruising	Fracture of Skull	Pupils	Neurological Signs	Pulse	Temperature °F.	Respiration	Findings at Operation: *Site of Hæmorrhage*	Findings at Operation: *Source of Hæmorrhage*	Result
11	16	M.	10 d.	—	Unconscious. Extreme restlessness	—	Left parietal	Normal reactions	Paresis of right arm	88	97	24	Left parietal	Vein (M.M.V.)	Recovery
12	13	M.	3 d.	—	Unconscious. Extreme restlessness	Bruising, œdema, right occipital region	Right parietal	Normal reactions	—	80	98	20	Left frontal (*contre-coup*)	Artery (branch of M.M.A.)	Recovery
13	32	M.	3 d.	—	Unconscious. Extreme restlessness	Bruising, œdema, right temporal region	Right squamous temporal	Right, fixed and dilated	Left hemiplegia	70	98	22	Right temporobasal	Artery (trunk of M.M.A.)	Recovery
14	27	M.	8 hr.	—	Rapidly deepening unconsciousness	Bruising, œdema, right mastoid region	Right parietooccipital	Right, fixed and dilated	Decerebrate rigidity	44	97	Cheyne-Stokes	Right occipitocerebellar	Right lateral sinus	Recovery
15	55	M.	5 d.	—	Gradual deepening of unconsciousness	—	Right parietal	Normal reactions	—	50	97	22	Right parietal	Diploic channel at fracture site	Recovery

the bleeding was coming from a torn lateral sinus. Following operation the patient remained extremely ill for two days before showing any sign of improvement, but he recovered completely.

Of the remaining 4 cases, 3 had lain unconscious for ten days, three days, and three days respectively. In each case a marked feature had been extreme restlessness—restlessness of such a degree that it suggested that the patients were reacting to severe pain—and this, no doubt, is the counterpart of the severe headache seen in the group of cases which have a latent period. All 4 cases were operated upon because of steadily progressing unconsciousness. *Case* 13 also developed a hemiplegia while under observation, and *Case* 11 developed paresis of the right arm. All the cases had fractures of the skull on radiography. *Case* 12 is a particularly important case, as this patient had remained unconscious and extremely restless for three days and then had begun to get more deeply unconscious. There was a large bruise in the right occipital region and a fracture in the right parietal bone visible on the radiographs. There were no localizing neurological signs and the reactions of both pupils were normal. The right side was explored first at the site of the bruise in the occipital region. This exploration proved to be negative extradurally and subdurally, and further negative explorations were made in the right temporal and frontal regions. On exploring the left frontal region, however, a very large extradural clot was located and removed through a wide exposure. This was the only *contre-coup* extradural hæmorrhage in this series, but it emphasizes the importance of exploring both sides of the head when a clot has been diagnosed on clinical grounds.

In each of these 5 cases an extremely large clot was found at operation and removed. All 5 patients made complete recoveries. Three of the cases were instances of convexity hæmorrhages from vessels of small calibre (*Cases* 11, 12, 15). One case had a massive occipito-cerebellar hæmorrhage (previously mentioned) and the fifth case was a hæmorrhage from the trunk of the middle meningeal artery (*Case* 13). This patient had been unconscious and restless for three days, when he developed a hemiplegia and a fixed, dilated, right pupil within the space of a few hours. He had a large right-sided temporobasal hæmorrhage from a torn middle meningeal artery. Presumably, this vessel, at some stage, had sealed off and then, following straining on the patient's part, the bleeding had recommenced, since he went off so rapidly.

PULSE, TEMPERATURE, AND RESPIRATION IN THE EXTRADURAL HÆMORRHAGES

From inspection of *Tables I, II*, it will be seen that only 7 of the 15 cases had pulse-rates below sixty beats per minute. When bradycardia occurs in association with unconsciousness it is a most valuable sign of cerebral compression and should immediately arouse the suspicion of intracranial hæmorrhage. However, it is by no means a constant finding in surface clot compression, and, indeed, it is a rare finding in all types of acute injury to the head. A rapid thready pulse is a most serious sign in intracranial hæmorrhage as it indicates failure of the cerebral circulation (e.g., *Case* 3).

The respiration rate in surface clot compressions at first is deep and fast—becoming deep and slow—until finally the breathing becomes irregular. Stertor and Cheyne-Stokes respiration are extremely dangerous signs—although 2 cases with Cheyne-Stokes respiration recovered completely after stormy convalescences, while 2 other cases with Cheyne-Stokes breathing succumbed.

PROCEDURE AND TECHNICAL DIFFICULTIES IN OPERATING FOR EXTRADURAL HÆMORRHAGE

The first consideration is whether or not to administer a general anæsthetic to the patient. In deeply unconscious patients local anæsthesia is the method of choice. In semi-comatose patients, and especially in children, one may have to resort to general, inhalational anæsthesia, administered through an intratracheal tube. In such cases the services of an expert anæsthetist are essential. Unskilled attempts at induction or intubation, resulting in an increase in the degree of cerebral congestion, will do untold harm and will surely lessen the chances of recovery.

Even with careful pre-operative study it may be difficult to diagnose with certainty the centre of the clot, and therefore it is well to adopt a routine in operating. In the presence of well-marked scalp bruising and positive radiographic evidence of fracture, a burr hole is made in close proximity to this area, as in the majority of cases extradural clots will be found immediately beneath the fracture site. In the absence of marked external injury, and when radiographs are not available or are negative for evidence of fracture, a burr hole is made in the anterior temporal region, since this is the site of maximum frequency of massive extradural bleeding. In either case, if clot is encountered extradurally, one is still in ignorance whether it extends in an anterior or a posterior direction. Therefore other burr holes will have to be made in order to delineate the area of clot, the scalp incisions being so placed that when they are united, an orthodox, well-vascularized scalp flap results. When the scalp flap has been reflected the bone is exposed by the technique of subperiosteal muscle slide (Rowbotham, 1945). The bone is then gently nibbled away with rongeurs, taking care not to compress further the already embarrassed brain. This manœuvre results in gradual cerebral decompression; with each pulsation of the brain the clot is extruded, and at this stage it is wise to pause for a few minutes to allow the cerebral circulation to readjust itself. A wide bony removal should always be made. Only when a sufficient amount of bone has been removed should an attempt be made to clear away the clot and find the bleeding point. The clot is removed gently, using a blunt dissector, and any small bleeding dural vessels are coagulated. Deep arterial spurting may be difficult to identify owing to adherence of small pieces of clot to the dura mater in the deeper reaches of the wound: this applies particularly to bleeding from the middle meningeal artery itself. For this reason, one should always be

prepared to give a blood transfusion, and in children a blood-drip should be started before the operation is begun.

Arterial or venous bleeding points on the dura mater can usually be arrested with the coagulation diathermy, but it may be necessary to clip the vessel with silver clips or to under-run it with fine silk on a dura needle. Rarely the foramen spinosum will have to be plugged with a matchstick, and in order to do this good lighting, preferably using a lighted retractor to hold up the temporal lobe, is essential. Diploic bleeding is arrested with Horsley's wax. In this series there was only one venous sinus tear—a large one—and bleeding in this instance was arrested with muscle grafts which were sutured across the tear.

The subdural space should always be explored, as on two occasions subdural hæmatomas have been found, and in one case a large subdural hygroma was released.

After ensuring absolute hæmostasis, the extradural plane should always be drained for twenty-four hours. In addition it is a wise precaution to explore the opposite side in two places, temporal and parietal, through burr holes. Although there were no bilateral extradural hæmorrhages in this series, they do occur, and even if there is not an extradural hæmorrhage on the opposite side there may very well be a subdural collection.

CAUSE OF DEATH IN THE THREE FATAL CASES

Post-mortem examinations were made on all three fatal cases. In one case, no further lesion could be demonstrated, and it is clear that death in this case had resulted from brain-stem compression which had not been relieved sufficiently early.

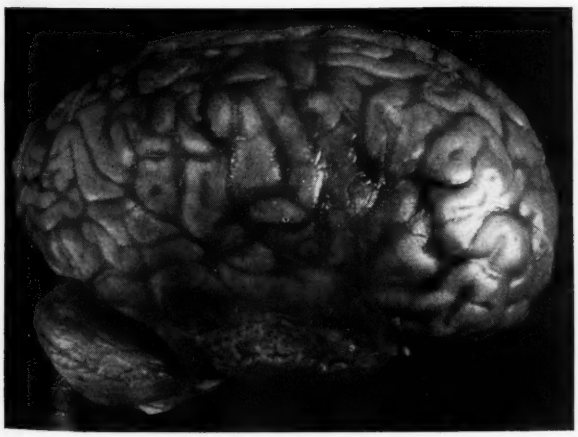

FIG. 281.—Photograph of brain in *Case* 9, showing large laceration of right temporal lobe, with surrounding area of contusion.

In another case, a small laceration of the temporal lobe was present which was responsible for a coincident subdural hæmatoma. But this patient had been well for twenty-four hours following the blow to his head, and it is highly probable that he would have recovered if operation had been performed earlier.

In *Case* 9 laceration of the right temporal lobe of the brain with surrounding contusion was found. It is possible that this was a contributory factor in the cause of death in this case (*Fig.* 281).

In none of the cases had there been any further extradural bleeding to account for the fatal issue.

ACUTE SUBDURAL HÆMORRHAGES NOT ASSOCIATED WITH SERIOUS INTRINSIC INJURY TO THE BRAIN

There are two types of acute subdural bleeding following trauma to the head. The first type is subdural bleeding in association with serious intrinsic injury to the brain. This is by far the most common type, for some degree of subdural bleeding is usual in severe injuries to the head. At the time of injury the patient suffers diffuse intrinsic damage to the brain, associated with local brain laceration. The bleeding comes from torn vessels in the areas of brain laceration. The clinical picture is that of immediate unconsciousness following injury, with superadded signs of rapidly advancing cerebral compression. Only very rarely is there a latent period. The subdural collection consists of a mixture of venous and arterial blood with cerebrospinal fluid, and for this reason a solid clot is seldom seen. The major problem here is that of the associated diffuse neuronal injury and experience has shown that surgery has little place in the treatment of this group of subdural hæmorrhages.

The second type of acute subdural bleeding is not associated with serious intrinsic injury to the brain (*Table III*). This is a rare type of hæmorrhage and, on clinical grounds, is indistinguishable from the classical extradural hæmorrhage (Whalley, 1948). A blow on the head is followed by a latent period (as in *Case* 16) or the blow results in a state of severe confusion (as in *Cases* 17 and 18). Thereafter the picture is one of steadily progressive cerebral compression. At operation there is no bleeding to be found in the extradural plane, but a massive, dark, gelatinous, well-localized blood-clot is present in the subdural space compressing the brain.

There were 3 such cases of acute localized subdural hæmatoma in the series of surface clot compressions. The first case (*Case* 16), a young girl, was hit on the head by a golf-ball. She experienced a momentary sensation of dizziness immediately following the blow, but she soon recovered and was able to go for a walk with her father. One hour later she complained of severe headache. Soon she felt sleepy and when she was put to bed she fell asleep at once. About an hour afterwards the parents had difficulty in rousing her. When seen at hospital she could be roused only with difficulty: she presented bilateral pyramidal signs which were more marked on the right side, together with an incomplete third-nerve palsy on the left side.

The second case (*Case* 17), a female adult, was seen six hours after she had slipped from the step of a trolley-bus, striking her head on the ground. The blow resulted in a state of moderate confusion which had persisted until admission. When examined she was almost maniacal with headache;

Table III.—Acute Localized Subdural Hæmorrhages

Case No.	Age	Sex	Period of Initial Unconsciousness	Latent Period	First Symptom or Sign of Cerebral Compression	External Scalp Bruising	Fracture of Skull	Pupils	Neurological Signs	Pulse	Temperature °F.	Respiration	Findings at Operation: Site of Hæmorrhage	Findings at Operation: Source of Hæmorrhage	Result
16	9	F.	Momentary dizziness	1 hr.	Severe headache	Transverse bruise at vertex of skull	No demonstrable fracture	Normal reactions	Ptosis left upper lid. Bilateral spasticity especially on right)	68	97	20	Left temporal	Not seen. ? ruptured cortical vein at tip of temporal lobe	Recovery
17	31	F.	6 hr.	—	Severe headache and increase of depth of unconsciousness	Bruising, left temporal region	Left temporal	Left, fixed and dilated	Bilateral spasticity	64	98	22	Left temporal	Not seen. ? ruptured cortical vein at tip of temporal lobe	Recovery
18	10	M.	12 hr.	—	Gradually deepening unconsciousness	Bruising, left frontal region	Right occipital bone	Left > right (sluggish reaction)	Ptosis (left), left hemiplegia	120	97	24	Left temporal	½-in. long laceration, left temporal lobe (superficial)	Recovery

she had a fixed, dilated, left pupil, and all four limbs were equally spastic, with bilateral extensor plantar responses.

The third case (*Case* 18), a young boy, was knocked down by a motor-bus and was seen twelve hours afterwards. He had been transferred to the Neurosurgical Unit because of gradually deepening unconsciousness in association with a developing left hemiplegia and an incomplete third-nerve palsy also on the left side.

It will be seen that the sequence of events in each case was that usually associated with extradural bleeding. At exploration in each case large, localized, subdural clots were exposed and evacuated. These clots were similar in every way to those found in the extradural series, with the exception that they were situated deep to the dura mater.

It proved impossible to suck out these clots; they were too gelatinous and tough, and a wide exposure had to be made in order to remove them. When sufficient bone had been removed the clots could be seen shining through the dura mater like large blue-black tumours with oval or rounded outlines. When the dura had been opened they could be gently broken up and removed. In each case after removal of the clot the underlying brain appeared to be œdematous and herniated gently through the site of dural opening, thus rendering closure of the dura impossible. Instead, the brain had to be covered with fascial grafts which were stitched to the dura mater.

The actual source of bleeding was only encountered in one case (*Case* 18)—a half-inch long superficial laceration of the temporal lobe. This was the only case in the whole series of extradural and subdural hæmorrhages which developed a hemiplegia on the same side as the clot, and due, no doubt, to the Kernohan-Woltmann crescent. In *Cases* 16 and 17 it was considered likely that the bleeding was coming from a ruptured cortical vein in the region of the tip of the temporal lobe, although no bleeding point was seen.

All 3 cases made complete and rapid recoveries within short spaces of time following operative removal of their clots, thus proving that surface clot compression had been the major factor in the production of the clinical picture.

SUMMARY AND DISCUSSION

The diagnosis of post-traumatic surface clot compression of the brain is essentially a clinical diagnosis. In cases where there has been a latent period, the development of severe headache in association with mental confusion, drowsiness, or giddiness should at once arouse suspicion of intracranial hæmorrhage—whether associated with bradycardia or not. The performance of lumbar puncture and spinal manometry, as an aid to diagnosis, should be unnecessary in this type of case, although it may prove of some value in the type of case which presents no latent period, and in which the patient has lain unconscious and restless for some days and where the neurological findings are minimal.

It has been shown that in cases where the bleeding vessel is large and situated low in the middle

fossa or in the occipito-cerebellar region the march of events is swift and the classical signs of extradural hæmorrhage may not be observed, because of rapid development of decerebrate rigidity, which follows on herniation of the temporal lobe through the hiatus tentorii.

Fixation and dilatation of one pupil to direct and consensual stimulation, while a valuable lateralizing sign, is usually a late sign, and it, too, indicates the development of a tentorial herniation which if not speedily relieved will surely lead to a fatal issue. Unilateral dilatation and fixation of a pupil in this series of cases occurred in 8 out of 18 cases, and clots were found on the same side as the fixed pupil in each case. In another case (*Case* 18) the left pupil was larger than the right, reacted sluggishly to direct light stimulation, and there was ptosis of the left upper eyelid: the clot in this case was situated on the left side of the brain.

External scalp bruising or hæmatoma formation is another valuable lateralizing sign. In 12 of the 18 cases in which external scalp bruising was in evidence, clots were found on the same side as the bruising. In another case (*Case* 12) with bruising in the right occipital region, the clot was situated in the left frontal region—the only *contre-coup* clot in the series. *Case* 16 had a transverse bruise at the vertex. The clot in this case—a subdural clot—was situated in the left temporal region.

Radiographs of the skull, if readily available, may provide helpful confirmatory lateralizing evidence, but they are not essential in diagnosis. It cannot be overemphasized that delay in operating, for any reason, is courting disaster.

When operating for the relief of cerebral compression due to intracranial hæmorrhage, local anæsthesia is the method of choice in deeply unconscious patients. In children and semicomatose patients one may have to resort to general intratracheal anæsthesia, but this must be administered by an expert anæsthetist. Unskilled general anæsthesia will surely lessen the chances of a successful outcome. At operation, a wide bony removal should always be made: all manipulations should be gentle, and no attempt should be made to clear away the clot until time has been allowed for the cerebral circulation to readjust itself. In children a blood transfusion should always be started before the operation. It is a wise procedure to inspect both sides of the head as a routine, for bilateral clots do occur, and even if there is no extradural clot on the opposite side, a subdural collection may be found.

All cases post-operatively require skilled and unremitting nursing attention and, in particular, those cases which have been and remain decerebrate for any length of time. A few of these patients are *in extremis* when first seen, and die in spite of all efforts, but a certain number of them can be saved and do recover completely (*Cases* 4, 14).

The results here presented show a recovery rate of 83 per cent in the combined extradural and subdural series. The 3 deaths occurred in the extradural series, and the recovery rate here is 80 per cent. This is in extremely close agreement with Rowbotham's (1945) figures: in a personal series of 33 cases of extradural hæmorrhage which he operated upon, there were only 7 deaths—a recovery rate of 79 per cent.

Since 1885, when Jacobson's classical paper concerning middle meningeal hæmorrhage was written, several important papers have appeared. From these it is clear that the mortality rate in massive extradural hæmorrhage is higher than is often thought. In Pringle's (1938) series of 71 cases of traumatic meningeal hæmorrhage there were 14 examples of unilateral extradural hæmorrhage, of which 8 died. In McKenzie's (1938) series, 9 out of 20 cases died, but 3 were not operated upon. Kennedy and Wortis (1936) studied 17 cases of extradural hæmorrhage which they encountered in six years. They found the mortality rate in the hands of the neurosurgical service to be 54·5 per cent; in the hands of the general service it was 75 per cent. Munro and Maltby (1941) studied 44 cases of extradural hæmorrhage, of which 38 were operated upon. Twenty of the cases subjected to operation died, as did the 6 unoperated cases, the mortality rate in the operated group being 52 per cent.

I believe that in the future, with improved skilled observation, earlier diagnosis, and prompt treatment, these results will be improved even further; for there can be no doubt that many of these massive surface clots are only brought to light in the post-mortem room.

All the patients were admitted to the Newcastle upon Tyne General Hospital under the care of Mr. G. F. Rowbotham. Through his courtesy I have had the privilege of treating these patients, and I would like to thank him for this, and for his advice at all times.

Dr. Kathleen Bell has anæsthetized the majority of the patients and the results are in no small measure due to her skill. I would like to thank her for her assistance.

Sister Gault and Sister Johnson have nursed these patients with devotion and understanding, and I gratefully acknowledge their help.

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DUPLICATIONS OF THE FOREGUT

SUPERIOR ACCESSORY LUNG (2 CASES); EPIPHRENIC ŒSOPHAGEAL DIVERTICULUM; INTRAPERICARDIAL TERATOID TUMOUR; AND ŒSOPHAGEAL CYST

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Teratology, the science of congenital malformations, was until recently a neglected branch of medicine. Few pathologists studied malformations or attempted to explain their morphogenesis, usually attempting to ascribe the abnormalities to a single aetiological factor, such as inflammatory processes or mechanical injury due to oligohydramnios and/or amniotic adhesions, or to genetic defects. In recent years, however, it has been shown that environmental factors can produce in the developing organism changes closely resembling or indistinguishable from genetic differences. The term 'phaenocopies' was introduced for such phenomena by R. Goldschmidt (1938). Such phaenocopies were produced by X rays in a variety of animals (Bardeen, 1911; Ancel and Vintemberger, 1925; Butler, 1936; and others). Aschenheim (1920) described the first case of multiple malformations due to the X-ray treatment of a myoma in the pregnant mother. The striking observations of Gregg (1945) and other Australian authors showed that rubella in the first few months of pregnancy almost invariably causes malformations in the fœtus. The protozoal infection in congenital toxoplasmosis may cause such malformations as hydrocephalus, microcephaly, microphthalmia (Wolf, Cowen, and Paige, 1939; and Sabin, 1942). Warkany (1947) and his associates produced malformations by nutritional deficiency in mother animals. These researches indicated a possible field in the prevention of congenital malformations, revived interest in their pathology, and made their study essential to 'antenatal pædiatrics' (Parsons, 1946).

To elucidate this problem not only the knowledge of the noxious agent but the time of its action and the mechanism producing individual malformations must be discovered. Thoracic malformations are not infrequently associated with abnormal arteries and bronchiectasis and are of great clinical importance. If the possible presence of such vascular abnormality is not borne in mind, dangerous bleeding may follow the division of diaphragmatic adhesions in the course of a lower lobectomy (Douglass, 1948). In this paper 5 apparently unrelated malformations, 2 cases of superior accessory lung, an epiphrenic diverticulum of the œsophagus, an œsophageal cyst, and an intrapericardial 'teratoid' tumour will be described and an attempt made to explain these malformations on the basis of a single morphogenetic principle.

CASE REPORTS

Case 1.—Bronchiectasis left lower lobe and inferior segment of lingula; accessory lung; aneurysm of basilar artery.

Ann Ph., aged 5 years, an only child, was admitted on Nov. 17, 1947, At 18 months a continuous productive cough, worse in winter, developed. She was well grown, not cyanosed, and had slight clubbing of the fingers. The tonsils were enlarged. Moist râles were heard at the base of the left lung. Bronchography showed bronchiectasis in the left lower lobe and in the lower segment of the lingula. In the upper part of the left chest a sharply outlined pyriform shadow was seen and thought to be an area of collapsed parenchyma (*Fig.* 282). The lipiodol filling in the upper half of the

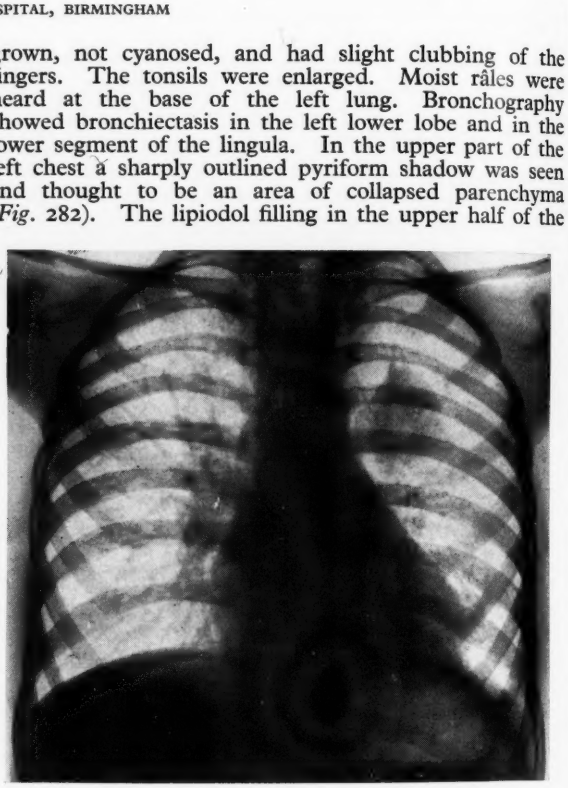

Fig. 282.—*Case* 1. Radiograph of the chest showing the pyriform shadow of the accessory lung.

left side of the thorax was poor, with none seen within the shadow. At bronchoscopy there was thick sputum in both bronchi and no abnormality was seen in the orifices of the lobar bronchi. The child was discharged on Dec. 16 and readmitted on Jan. 27, 1948. An X-ray examination showed the lungs almost free from lipiodol and the pyriform shadow unchanged.

At Operation (Feb. 11).—Left lower lobectomy and resection of the lower segment of the lingula were performed. Few adhesions were present and none to the upper lobe. The lower lobe was atelectatic, and from an unusually large lingula a bronchiectatic lower segment was removed. The immediate post-operative condition was satisfactory, but next day the child deteriorated, becoming unconscious and the breathing stertorous. The left arm and leg were spastic and an extensor plantar reflex was present on both sides. The diagnosis (later proved incorrect) of cerebral air embolism was made, and she died at 8.40 p.m. on Feb. 12.

Pathology.—

Autopsy.—The cause of death was cerebral thrombosis starting in an aneurysm of the basilar artery, with areas of necrosis in the pontine nuclei and in the white matter near the dentate nucleus in the cerebellum. One ball hæmorrhage and a few ring hæmorrhages were seen in one cerebral peduncle. The thrombosis had extended into the posterior cerebral arteries.

There was a considerable amount of mucopus in the tracheobronchial tree: the left upper lobe was unexpanded

in the left pneumothorax space and large areas of the right lung were atelectatic. The left lung showed a greatly hypertrophied upper lingula segment, about half the size of the remainder of the upper lobe, to which it was connected by a thin bridge of lung tissue at the posterior end of a deep fissure. The lymphatic glands in the hilum were slightly enlarged.

The Accessory Lung (*Fig.* 283).—In the upper part of the left pleural cavity was a soft mass (4 × 3 × 1·5 mm.) covered by pleura and attached to the upper lobe by a thin band of connective tissue, within which lay a branch of the left pulmonary artery and vein. In the mass no cartilage could be felt. The sectioned lump showed a shell of soft red tissue 2·6 mm. in thickness, lined by smooth white membrane which enclosed a thick slimy white fluid, a smear from which showed epithelial cells and lymphocytes, but no organisms. Lipiodol, injected into the left pulmonary artery, was shown by radiography to have passed into the 'accessory lung', but when injected into the left main bronchus it failed to enter the mass.

Histology (Serial sections; hæmatoxylin-eosin, azan, Verhoeff's or Weigert's elastic tissue stain with van Gieson and Laidlow's reticulum stain): The wall was lined by a ciliated columnar epithelium with a layer of basal cells, a basement membrane, and a muscular coat (*Fig.* 284). In two places plates of cartilages were present. Mucous glands were also seen, some of them

FIG. 283.—*Case* 1. Thoracic organs. The arrow points to the opened cyst of the accessory lung.

in continuity with the lining epithelium. Spurs of about 9·5 mm. length, projecting into the cavity of the cyst, when followed in serial sections proved to be folds with a broad base, rapidly tapering towards the lumen. Their lining was identical with that of the whole cyst, their core containing elastic fibres, capillaries, and loose connective tissue. Apart from the spurs there were pouches, the largest 1·3 mm. in diameter, without any side branches. The wall of the cyst contained many wide bronchi, lined by columnar or by cubical epithelium. They contained an abundance of elastic fibres in their walls, but rather scanty muscle-fibres. Some of these bronchi continued almost without change in diameter into tubes with the characters of alveolar ducts, no respiratory bronchioli being interposed between the two types of tubes. In other parts wide bronchioli were

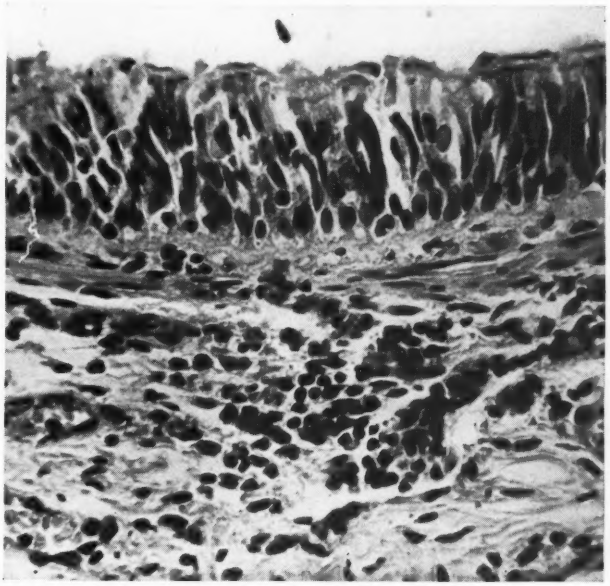

FIG. 284.—*Case* 1. Section of the wall of the cyst H. and E. (× 435.)

connected with wide alveolar ducts by a narrow and short middle piece which had an incomplete lining of low cubical or flat epithelium resembling alveolar ducts. In many of the bronchi and bronchioli papilliform proliferation of the epithelium and multiple pouching was seen. Many large solid clusters of epithelium were seen attached to the lining epithelium of a bronchiolus or bronchus, which when followed in serial sections showed a tiny central cavity communicating by a narrow lumen with the corresponding bronchial lumen. Most of the bronchial branching appeared to originate in such epithelial proliferations with secondary canalization from the bronchial lumen. The parenchyma of the cyst wall consisted largely of alveolar ducts with a few rudimentary alveoli, occasionally so small that a single alveolar macrophage filled the whole lumen. The bronchi, bronchioli, and alveolar ducts were mainly filled with a pale basophilic granular material, large pulmonary macrophages, and occasionally ball-like masses of deeply basophil granular material, but occasionally occupied by intact red blood-corpuscles. The septa between the alveolar ducts showed distended capillaries and histiocytes. The interstitial tissue resembled embryonic mesenchyma with widely scattered stellate and fusiform cells. The arteries had the normal structure of pulmonary arteries. The pedicle consisted of loose connective tissue with a few small lymph-nodes, numerous bundles of nerve-fibres, and an artery 0·83 mm. in diameter. No bronchi or any other elements of lung tissue were seen in the pedicle.

The Lobectomy Specimen.—In the atelectatic, firm lower lobe were groups of dilated bronchi in some areas but not in all. Both in the lobe and the removed part of the lingula were areas of collapse and fibrosis. Histological examination showed wide bronchial lumina up to 6 mm. in diameter, the lining being a ciliated columnar epithelium; of the inflammatory cells the plasma-cells

predominated. In some areas there was marked muscle hypertrophy, with elastic fibres, scarce or absent, though in some there were many such fragmented fibres—a comment equally applicable to some of the alveolar ducts and inter-alveolar septa. Lymphadenoid hyperplasia was marked. In large areas the parenchyma was replaced by fibrous tissue within which tubuli lined by cubical epithelium, an occasional island of cartilage, or plain muscle and elastic fibres were seen.

Case 2.—**Accessory tracheal lung** (*Fig.* 285).

D. P., male infant aged 3 days at the time of death. The mother had a threatened abortion at three months' gestation. The baby was prematurely born, with a

FIG. 285.—*Case* 2. Thoracic organs. The probe is passed through the abnormal opening in the trachea. Arrow (*a*) points to the accessory lung, arrow (*b*) to the upper lobe of the right lung.

birth weight of 3 lb. 12 oz. and a length of 17 in. The condition at birth was good, but on the third day he became cyanosed and dehydrated and was admitted to the Carnegie Premature Unit *in extremis* with Cheyne-Stoke's type of respiration, dying soon after admission.

AT AUTOPSY.—On post-mortem examination an abnormal opening was seen on the right side of the trachea 7 mm. above the bifurcation. In the right pleural cavity there was, apart from a normally configurated lung, a mass of lung tissue (25 × 12 × 5 mm.) covered by a smooth and lustrous serous membrane, situated above and medial to the apex of the right upper lobe, between it and the trachea. It was closely attached to the trachea by a narrow band of connective tissue and linked with the upper lobe of the right lung by a thin membrane 9 mm. in length, 4 mm. in width, and a fraction of a millimetre in thickness. Within this membrane, and passing from the upper lobe into the accessory lung, there was a forking artery, a vein, and between these two, a whitish cord or tube. A probe passed from the abnormal opening in the trachea went into the upper lobe of the right lung. No abnormal blood-vessels, either from the aorta or from the subclavian artery, were found supplying the accessory lung.

The other main post-mortem findings were: a horseshoe kidney, a right congenital hydro-ureter, submucous hæmorrhages of the duodenum and of the urinary bladder, tracheobronchitis, and hæmorrhagic bronchopneumonia.

Histological Findings.—The caudal part of the trachea, together with the accessory lung and a part of the right upper lobe, was cut in serial sections and stained with a variety of stains. Study of these sections showed the accessory bronchus having a diameter of 1·5 mm. and passing at an angle of approximately 45° downwards and to the left in a plane which slightly deviated anteriorly from that of the trachea; 5·5 mm. below its origin from the trachea the bronchus divided dichotomically, the rostral branch supplying the accessory lung, the caudal forming an accessory bronchus for the upper lobe (*Fig.* 286). The arterial supply of the accessory lung was

FIG. 286.—*Case* 2. Section through the trachea, the abnormal bronchial tube with its dichotomous branching, the accessory lung, and part of the upper lobe of right lung. Arrow (*a*) points to the abnormal bronchus, arrow (*b*) to the accessory lung. Weigert's stain for elastic fibres with van Gieson counterstain. (× 2.)

from the pulmonary artery and no branch from the systemic circulation was seen passing to it. The bronchus for the accessory lung passed through the centre of the latter and divided further dichotomously. The pulmonary parenchyma within the accessory lung was incompletely collapsed and numerous septal cells were present in the air spaces.

Case 3.—**Left lower-lobe bronchiectasis; congenital epiphrenal œsophageal diverticulum; aberrant artery.**

P. T., a girl 6 years of age, had suffered from "recurrent bronchitis and pneumonia" since four months of age. She had been in poor general health and undersized all her life. At the age of 2 years a diagnosis of bronchiectasis in the left lower lobe was made on clinical and radiological evidence. In February, 1947, she was admitted to the hospital, and on the basis of radiological findings (*Fig.* 287) a diagnosis of bronchiectasis associated with a lung abscess was made. After pre-operative treatment she was submitted to operation on May 16, 1947.

AT OPERATION.—The upper lobe was healthy, but the lower lobe showed areas of gross atelectasis and was densely adherent to the parietal pleura, especially over the diaphragm. The adhesions were divided carefully and a large artery was seen coming through the diaphragm as that muscle crossed the aorta: it was divided between

artery forceps and the cut lumen of the artery showed the creamy yellow colour of elastic arteries. As the artery was followed towards the lower lobe it divided over a 'cyst' situated partly in the mediastinum. Further investigation of the 'cyst' (thought pre-operatively adherent to the epiphrenic diverticulum. The œsophagus was mobilized and a clamp placed across the mouth of the diverticulum, which was resected together with the lower lobe (*Fig.* 288). The œsophagus was sewn over by two layers of interrupted thread sutures.

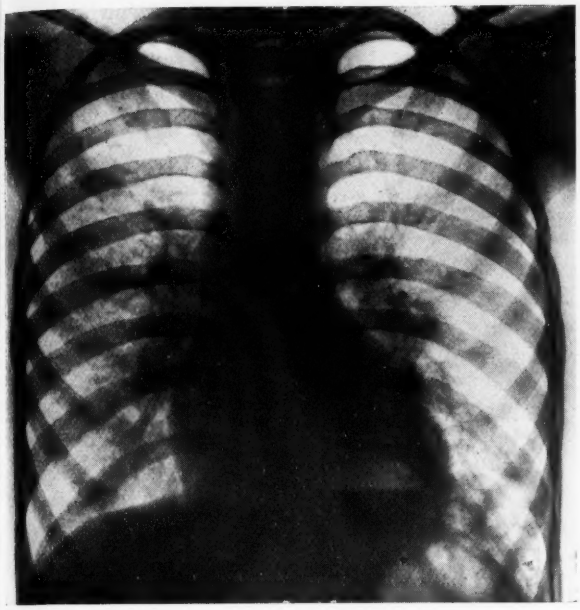

FIG. 287.—*Case* 3. Radiograph of the chest showing the epiphrenic œsophageal diverticulum with a fluid level seen through the heart shadow, and cystic bronchiectasis of the left lower lobe.

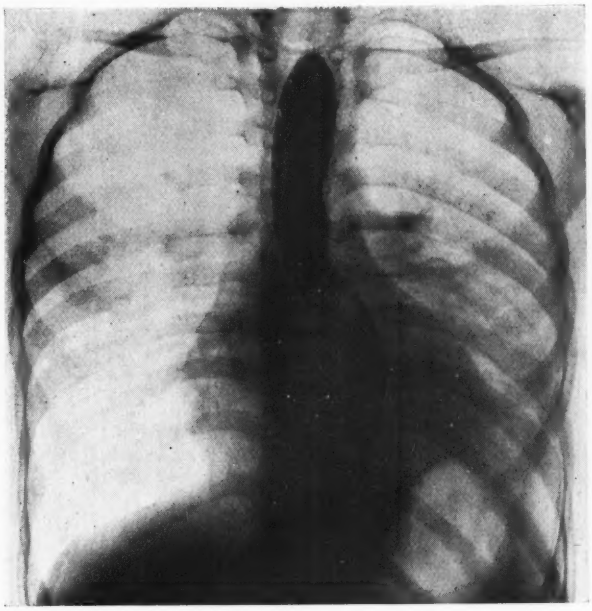

FIG. 289.—*Case* 3. A barium swallow after operation, showing deviation of the œsophagus to the left.

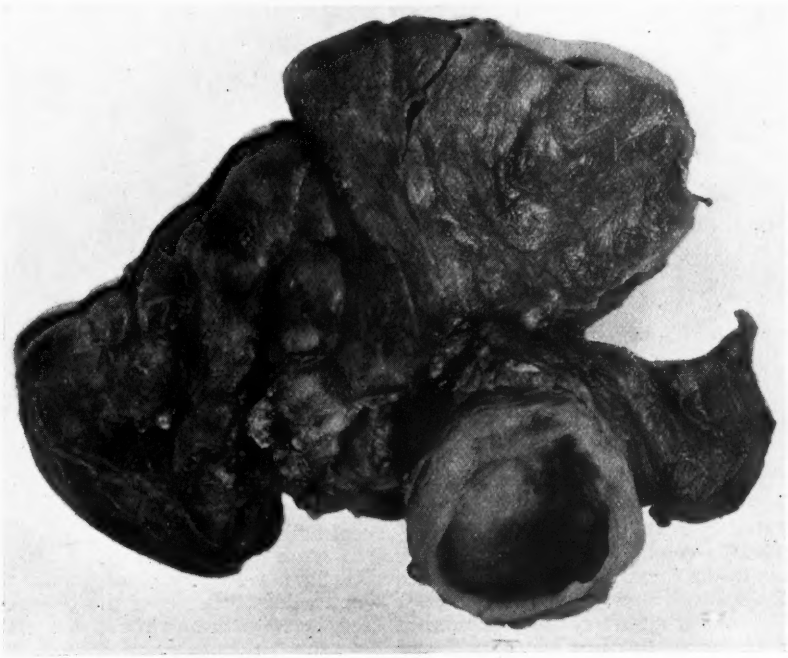

FIG. 288.—*Case* 3. Operation specimen showing left lower lobe, the œsophageal diverticulum, and, at its lower margin, the cut end of the abnormal artery.

to be a lung cyst or abscess) disclosed a large diverticulum of the œsophagus. The pulmonary artery and vein to the lower lobe were then isolated and divided between ligatures. The bronchus was then divided, the proximal end being closed by interrupted linen thread sutures and a pedicled pleural flap. The lobe remained firmly

Post-operative progress was satisfactory apart from the development of a small empyema (Gram-negative bacilli) which disappeared completely after several aspirations. The child has recovered well and is symptom-free. *Fig.* 289 is a radiograph of the œsophagus after operation, showing some deviation to the left.

THE PATHOLOGY OF THE EXCISED LOBE AND DIVERTICULUM.—The operation specimen consisted of the lower lobe of left lung; attached to the pulmonary ligament and the lower medial part of the lobe there was a pouch forming two-thirds of a sphere and measuring 32 mm. in diameter. The pouch was lined by a smooth yellowish-pink membrane with a number of pinhead to over

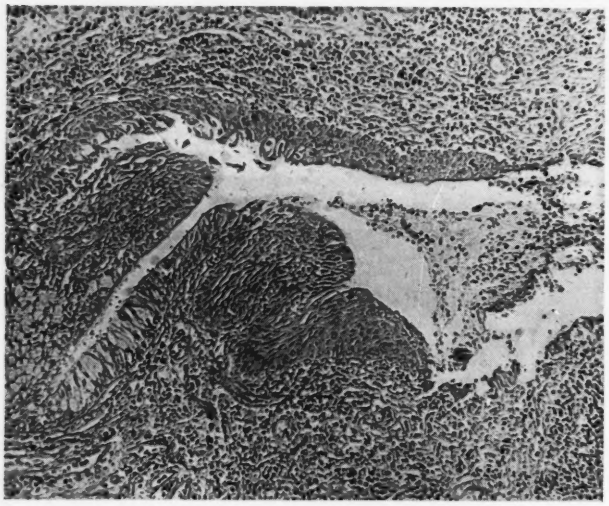

FIG. 290.—*Case* 3. Ectatic bronchus of the lower lobe of left lung with circumscribed squamous epithelial metaplasia. H.E. (× 90.)

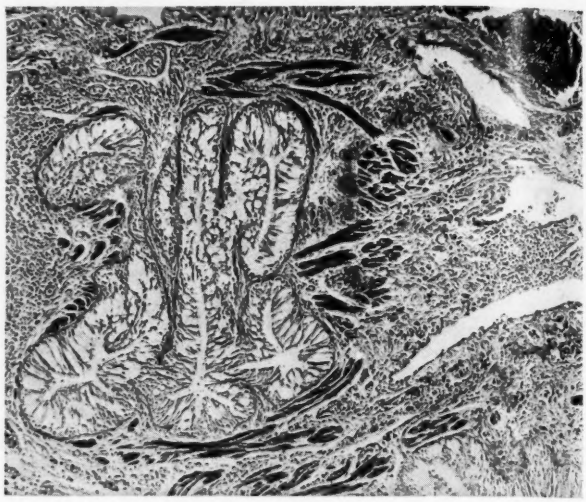

FIG. 291.—*Case* 3. Bronchus of left lower lobe. Hyperplasia and hypertrophy of the muscular coat. Azan. (× 72.)

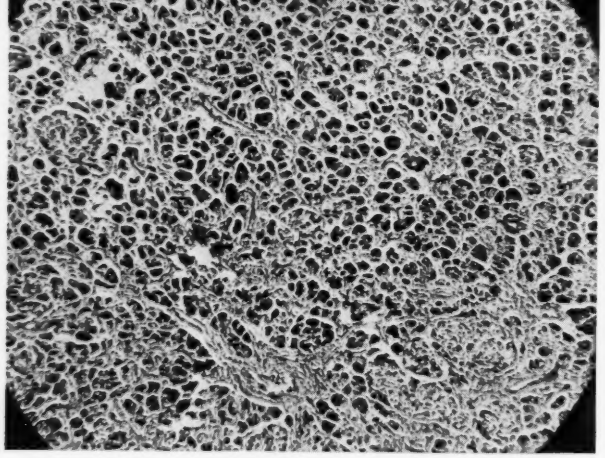

FIG. 292.—*Case* 3. Pancreatic tissue attached to the wall of the œsophageal diverticulum. H.E. (× 72.)

FIG. 293.—*Case* 3. Cross-section of the abnormal artery supplying the œsophageal diverticulum. Weigert's stain for elastic fibres with van Gieson counterstain. (× 90.)

lentil-sized spots of dark-red colour and several streak-shaped patches of similar colour (dilated veins). The wall of the pouch was in the thinnest part 2 mm., in the thickest 6 mm., in thickness. At the postero-inferior part of the pouch a stump of an artery about 2 mm. in length was seen. This artery supplied the pouch, and no branches to the lung were seen on naked-eye examination. The pouch was connected with the lung by a layer of loose connective tissue.

The cranial one-quarter of the lobe consisted of collapsed lung parenchyma. The caudal three-quarters showed on sections dilated bronchi and cavities 2–5 mm. in diameter with trabeculated walls. The tissue between these cavities was fleshy in some parts, firm and fibrous in others, and spongy in others.

Histology.—Sections showed numerous wide cavities lined by ciliated columnar epithelium, many of the cells being distended with mucus. This was particularly marked in the larger bronchi with cartilage in their walls. In such bronchi hypersecretion was also evident in the mucous glands. In some areas there was a metaplasia of the lining epithelium into stratified squamous epithelium (*Fig.* 290). Such metaplasia was confined to small areas, and patches of stratified squamous epithelium were seen surrounded by columnar ciliated epithelium. Some of the bronchi showed proliferation of the epithelium without metaplasia, and here and there a bronchial lumen was obliterated by shed-off epithelial cells undergoing necrotic changes. The elastic fibres of the bronchial walls were in some areas absent, while in others clumps of curled-up, fragmented fibres were seen. Muscle-fibres were also absent along large areas of bronchial walls, while in other parts there was a definite hypertrophy of the muscular coat (*Fig.* 291). The bronchial walls and the peribronchial tissue were densely infiltrated with lymphocytes, plasma cells, monocytes, and a rather large number of eosinophil

cells. The latter were either polymorphonuclears or myelocytes. There was also a circumscribed lymphadenoid hyperplasia with lymph-follicles showing large germinal centres. Either in close vicinity to dilated bronchi or separated by areas of cellular infiltration there were numerous tortuous tubuli lined by cubical epithelium and resembling in their structure terminal bronchioli. No alveolar ducts and alveoli were seen in such areas. In other parts the lung parenchyma was entirely replaced by fibrous tissue. In some areas, mainly in subpleural regions, there were very wide alveolar ducts and alveoli. Here and there remnants of interalveolar septa were seen projecting like short spurs into the wide air spaces. In some areas air spaces resembling alveolar ducts were separated by connective tissue in which there were numerous plain muscle-fibres without any regular arrangement. Elsewhere the absence of elastic fibres in the walls of alveolar ducts and interalveolar septa was a conspicuous feature. Areas of collapse were frequent and often extensive.

In the band connecting the lung with the œsophageal diverticulum there was an area of pancreatic tissue measuring 12 × 7 mm. in sections which apparently passed through its centre. This was normal in structure, with acini, lobuli, ducts, and islands of Langerhans (*Fig.* 292). The secretory cells contained eosinophil zymogen granules. This pancreatic tissue extended close to the pulmonary tissue, from which it was separated by connective tissue in some areas only 50–100 μ in thickness. In one area a large group of mucous glands was closely attached to the pancreatic tissue. The pouch was lined by stratified squamous epithelium and its wall had the normal structure of an œsophageal wall. In the outer coat there were numerous acinous serous glands and clusters of lymphocytes. Small groups of acini were also seen within the lymphadenoid tissue.

The artery supplying the diverticulum had the structure of elastic arteries and branches with a similar structure were seen around the pancreatic tissue and penetrating towards the lung tissue (*Fig.* 293).

FIG. 294.—*Case* 4. Heart with the intrapericardial ' tumour '.

Case 4.—**Intrapericardial ' tumour '.**

Baby P. The mother had had hydramnios. The membranes ruptured at 28 weeks' gestation, and the premature baby, after a few shallow breaths, died. Body length 15½ in., body-weight 3 lb. 3 oz.

POST-MORTEM FINDINGS.—Marked anasarca of face, scalp, and neck. No œdema of legs. Almost the whole thoracic cavity was filled by an enormously distended pericardial sac through which a bluish fluid was seen. The lungs were concealed in the costovertebral grooves.

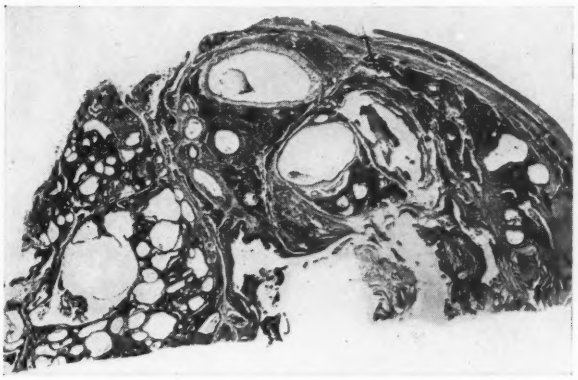

FIG. 295.—*Case* 4. Section of the ' tumour '. H. and E. (× 3·75.)

The pericardial sac was filled with approximately 3 oz. of thin, blood-stained fluid. At the base of the heart, attached to the pericardial reflexion, there was a globar, well-encapsulated tumour, more than twice as large as

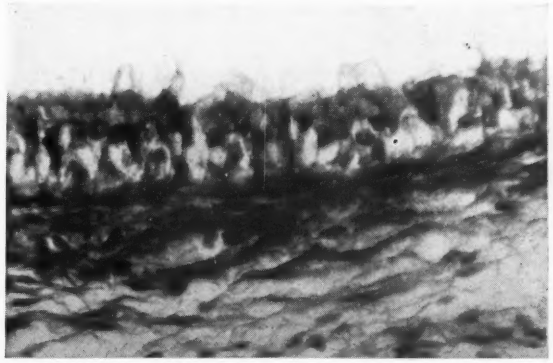

FIG. 296.—*Case* 4. Lining epithelium of one of the cystic spaces. H. and E. (× 390.)

the heart. It was 36 × 30 × 28 mm. in size. On the cut surface the tumour had a spongy appearance (*Fig.* 294), with numerous pin-head, millet-, lentil-, and small-pea-sized cystic spaces containing yellow fluid. In one area the ' tumour ' tissue was not spongy, and firmer in consistency. This area was greyish-white in colour while the septa in the spongy part were yellowish-pink. There were about 150 ml. of clear straw-coloured fluid in the abdominal cavity. Otherwise there were no relevant findings. The heart was not dissected.

Histology.—There were numerous spaces in the ' tumour ' lined by ciliated columnar epithelium (*Figs.* 295, 296), others lined by a single layer of cubical or flat epithelium, others lined by stratified epithelium. Plain muscle-fibres were seen in the walls of cystic spaces. In one area a plate of cartilage was seen in close relation to a cystic space. Between the cysts groups of acini were seen closely resembling those of a pancreas (*Fig.* 297), some with zymogen granules. Occasionally a structure was seen resembling an island of Langerhans. In some areas a ' cellular infiltration ' was seen—apparently foci of extramedullary hæmatopoiesis. Occasionally fusiform cells were seen with dark nuclei, sometimes lining

a narrow lumen. There was a marked extramedullary hæmopoiesis in the liver and spleen, otherwise no significant findings.

FIG. 297.—*Case* 4. Section of the 'tumour', showing cystic spaces and pancreatic tissue. H. and E. (× 60.)

Case 5.—**Œsophageal cyst.**

R. T. P., age 30 hours. Born after 34 weeks' gestation. There was a rapid second stage of labour. The condition at birth was quite good, but deteriorated rapidly. The baby developed grunting respiration and had cyanotic attacks.

AT AUTOPSY.—On post-mortem examination frothy seromucous, slightly blood-stained exudate was found in the trachea, about 10 ml. of clear fluid in each pleural cavity, and numerous flame-shaped subepicardial

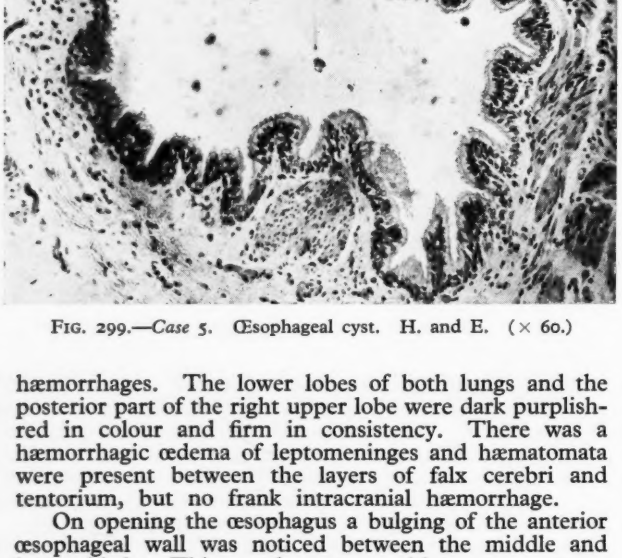

FIG. 299.—*Case* 5. Œsophageal cyst. H. and E. (× 60.)

hæmorrhages. The lower lobes of both lungs and the posterior part of the right upper lobe were dark purplish-red in colour and firm in consistency. There was a hæmorrhagic œdema of leptomeninges and hæmatomata were present between the layers of falx cerebri and tentorium, but no frank intracranial hæmorrhage.

On opening the œsophagus a bulging of the anterior œsophageal wall was noticed between the middle and lower thirds. This was due to an ovoid cyst 10 × 7 × 5 mm. in size, with a thin translucent wall, apparently containing fluid and firmly attached to the anterior aspect of the œsophageal wall (*Fig.* 298).

Histology.—In sections the maximum diameters of the cyst were 5 × 3 mm. The cyst was lined by ciliated columnar epithelium (*Fig.* 299). In some places there

FIG. 298.—*Case* 5. Œsophagus with the cyst in its wall.

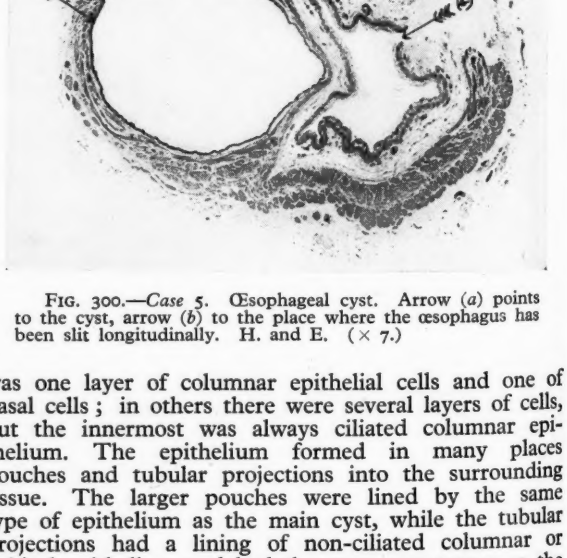

FIG. 300.—*Case* 5. Œsophageal cyst. Arrow (*a*) points to the cyst, arrow (*b*) to the place where the œsophagus has been slit longitudinally. H. and E. (× 7.)

was one layer of columnar epithelial cells and one of basal cells; in others there were several layers of cells, but the innermost was always ciliated columnar epithelium. The epithelium formed in many places pouches and tubular projections into the surrounding tissue. The larger pouches were lined by the same type of epithelium as the main cyst, while the tubular projections had a lining of non-ciliated columnar or cubical epithelium and had the same appearance as the ducts of mucous glands in the œsophageal wall. The epithelium rested on a basement membrane which was surrounded by a narrow layer of areolar tissue with few

elastic fibres. This areolar tissue was followed by plentiful plain muscle-fibres. Where the cyst was fused with the wall of the œsophagus these muscle-fibres were continuous with the outer muscular coat of the œsophagus. The free aspect of the cyst had two layers of the muscle-fibres, the inner having the same direction as the longitudinal fibres of the œsophagus, the outer at 90° to the former (*Fig.* 300). Between the muscle-fibres there were rather coarse widely scattered elastic fibres. The epithelial pouches penetrated deeply into the œsophageal wall and were surrounded by a thick layer of collagenous and elastic fibres. No cartilage was seen in the wall of the cyst.

DISCUSSION

The first case of this series is in several respects a unique example of an accessory lung. An accessory lung as defined by H. Müller (1928) is an "organ-like formation, consisting of pulmonary tissue and separated from the rest of the lung". He reviewed 26 examples of accessory lung described up to 1928. Two only were upper or 'tracheal' accessory lungs, the others being of the lower type, mainly on the left side, either thoracic, in the angle between aorta, œsophagus, diaphragm, and lower lobe of left lung, or abdominal. Several were associated with a congenital diaphragmatic hernia. In 1947 Valle and White found in the literature 37 cases of lower accessory lung, but added no further example of the rare superior type. The superior type is also called 'tracheal accessory lung' because in both described cases, that of Herxheimer and that of Müller, the accessory lung was connected by a separate bronchial tube with the trachea above the bifurcation as in *Case* 2. The arterial supply in Herxheimer's case (1901) (re-investigated by Bert and Fischer, 1911) was from a branch of the subclavian artery, in Müller's case apparently from small branches of the descending aorta. Thus, like the inferior type, these two superior accessory lungs received their blood from the systemic circulation.

In our *Case* 1 the accessory lung was completely separated from the tracheobronchial tract. The situation of the stalk suggested that the fœtal connexion was with the left main bronchus. In contradistinction to all cases of accessory lung which have been examined for the arterial supply, this one was supplied by a branch of the left pulmonary artery. Its structure, as in most recorded examples, was characterized by the formation of a bronchial cyst, with marked defective differentiation of the bronchial elements. The associated bronchiectasis in the left lung, present in this case and also in that of the œsophageal diverticulum (*Case* 3), may be significant. Although both the bronchiectases were infected and the histological differentiation between congenital and acquired bronchiectasis equivocal, some of the findings favour a congenital malformation, such as the absence or scarcity of elastic fibres, which are the most resistant elements in destructive processes, and the presence of layers of hypertrophic plain muscle-fibres around the whole circumference of some dilated bronchi.

Case 2 is a tracheal accessory lung like those of Herxheimer and of Müller, but differs in that a branch of the abnormal bronchial tube passes through a band of connective tissue to the right upper lobe and that the arterial blood-supply is from the pulmonary artery.

Two explanations of the morphogenesis of accessory lungs were for many years subjects of discussion. The first was that of Rektorzik (1861), who suggested a "fractionation" or "sequestration" of the pulmonary primordium by pleural adhesions. The second was Eppinger's (1904) hypothesis of supernumerary budding. Somewhat between these two is the explanation given by Cockayne and Gladstone (1918): "Accessory lungs are derived from the embryonic tissue of the 'pulmonary groove' or of the 'lung buds.' A portion of the embryonic pulmonary tissue, after having become adherent to the neighbouring part or organ, from which it draws its blood-supply secondarily, may be separated from the parent tissue and grow independently."

The sequestration theory until recently in disfavour was revived by Pryce (1946, 1947), who puts the whole emphasis on the abnormal blood-supply and assumes a sequestration by this abnormal vessel at any stage of the embryonic development from the first formation of the lung buds (end of fourth week) onwards. There are, however, several reasons why we doubt Pryce's theory:—

1. In our 2 cases the accessory lungs are supplied by a branch of the pulmonary artery and not by an abnormal vessel arising from the aorta.

2. Hammar (1904) described two isolated epithelial vesicles with the structure of bronchi in an embryo 11·7 mm. in length. Blood-vessels were absent from the 'accessory lung' as well as in the stalk.

3. The vascular system plays a comparatively small role in the early embryonic development. There are many branches supplied by the paired aortæ to the archentron, of which "the cœliac axis, superior and inferior mesenteric arteries are the sole survivors" (Sir Arthur Keith, 1948). The whole developmental history of the vascular system suggests that development or regression of vascular branches are determined by the neighbouring organs, which they later supply with blood. "It appears as if misplaced germs attracted the blood-vessels from the neighbourhood" (Bert and Fischer, 1911). Considering the structure of the abnormal arteries, which resembles that of pulmonary artery and to which Pryce attaches so much importance, it has to be realized that the detailed architecture of blood-vessels is mainly determined by hydrodynamic factors (Oppel and Roux, 1910; Huxley and de Beer, 1934).

It is also worthy of note that on the basis of purely morphological investigation Beneke (1905) anticipated nineteen years before Spemann's classical work (1938) some forces of dependent development. Discussing the conspicuous width of the abnormal arteries supplying accessory lungs, he remarks: "The misplaced pulmonary parenchyma is apparently able to form the characteristic pulmonary blood-vessels out of any vascular system, an obvious evidence of the determining influence of the pulmonary parenchyma on the stroma."

4. There is reason to believe that ciliated epithelial cysts embedded in the wall of the œsophagus are of the same nature as accessory lungs. In our

Case 5 there was no separate artery supplying the cyst.

Supernumerary buds are, on the other hand, common in the normally developing gut and its derivatives (Lewis and Thyng, 1908; Lacquesse, 1895). The regression of such buds is determined by the factors that regulate embryonic growth and differentiation—namely, the presence of intact organizers liberating evocators which determine the growth of an organ, and the "modulators" (Waddington), or "eidogens" (Needham), which further differentiate the product of an evocator stimulus. That such 'embryonic hormones' may have also an inhibitory effect is substantiated by Needham: "One genuine inhibiting effect, at any rate, is the influence exerted by the prechordal plate (anterior end of the notochord) on the presumptive eye-districts lying above. After a great deal of confusions about the origin of cyclopia, Andelman succeeded in showing that normally the two eye-districts overlap and that the development of this intermediate material into eye is inhibited by the prechordal plate. If this is interfered with, whether mechanically or by action of lithium, cyclopia results." (*See also* Lehmann.)

To indicate that damage to an organizer may result in the development of an accessory bud as well as in suppression of the development of an organ, Sir Arthur Keith refers to old observations of John Hunter, and concludes: ". . . if the dominant centre of growth loses control, as the result of being subjected to injurious conditions, then it may be replaced by two separate centres. If the injury is so great that the growing centre is killed or starved then the part fails to develop."

The work of experimental embryologists provides also an explanation of the abnormal differentiation of accessory lungs. In most cases, as in our *Case* 1, the accessory lung was not normal in structure, not a simple 'miniature lung', but showed cyst formation, excessive bronchial proliferation, with defective differentiation of air sacs. A similar observation may be made in hypoplastic lungs, whether the hypoplasia be due to intrinsic factors or to mechanical factors such as diaphragmatic hernia or intra-uterine cardiac hypertrophy (2 cases of the former condition and 1 of the latter were studied by the authors). A variety of observations lead Child (1936) to the assumption of metabolic gradients in embryonic development. This conception has been recently broadened by Dalcqu and Pastels (1938) and by Brachet (1947). According to them chemical substances diffuse along a gradient from at least two centres and it is the product of the concentration of substances from one and the other centre which determines the fate of a territory—its "morphogenetic potential". It is obvious that a growing accessory bud, displaced within the interacting metabolic gradients, will deviate from the differentiation of the normal organ. In *Case* 2, however, where the accessory lung was supplied by a separate abnormal bronchial tube from the trachea and where a second branch of this abnormal bronchus provided for a continuity with the right upper lobe, the differentiation of the pulmonary tissue was normal.

We assume, therefore, that as a result of a noxious agent, acting on the organizer for the development of lungs in the way of an alteration of its metabolic rate, an accessory bud has been formed and did not regress but, growing in a gradient different from that in which normal lung develops, formed an accessory lung with defective differentiation. The abnormal bud being derived presumably from the primordium of the left main bronchus in the first case and from the laryngotracheal ridge in the second, the teratogenetic determination period is to be placed between the end of the fourth and the beginning of the fifth week of embryonic life (Arey, 1943; Patten, 1947).

Case 4 will be discussed next. It is almost identical with one recently described by Willis (1946) and very similar to the four other 'teratoid' intrapericardial tumours (Joel, 1890; Grimm, 1927; Jellen and Fischer, 1936; Gebauer, 1942–3). We cannot agree with the interpretation of such 'tumours' as teratomata. They do not conform with Willis's (1948) own definition of teratoma. There is no evidence of progressive, autonomous, and inco-ordinated growth and they do not consist of tissues "utterly foreign to that part". They are situated in a region closely related to the foregut and contain no elements foreign to the intestinal primordium and mesoderm. This applies equally to cysts lined by ciliated epithelium and to the islands of cartilage as to the pancreatic tissue, which is seen as a derivation of the foregut also in *Case* 3. It is interesting that pancreatic tissue found in two of the present series is as frequent as aberrant tissue in such developmental abnormalities as Meckel's diverticulum. The development of pancreatic tissue from the primitive endoderm is apparently a form of self differentiation of which the whole fore- and most of the mid-gut are capable and its limitation to one area is due to normal inhibitory influences. The separation of an accessory bud or cell group from the foregut and its inclusion within the pericardial sac must have taken place in a comparative early stage—when the respiratory primordium is just formed, the pericardial sac not completely closed, and the broad dorsal mesocardium (arterial mesocardium) permits the inclusion of an accessory bud into the pericardial cavity. This would correspond to about 2·5–3 mm. crown-rump length, about the middle of the fourth week, and about 10–14 somites (Arey, 1943; Patten, 1947).

Albrecht (1904) has introduced the term 'hamartoma' for tumour-like tissue malformations and 'choristoma' for tumour-like malformations due to segregation of parts of embryonic primordia. According to this classification the 'tumour' in *Case* 4 is to be considered as a choristoma and not a true neoplasm. One of us has recently removed such a 'tumour' from the fissure between the right middle and right upper lobe together with the bronchiectatic middle lobe.

Case 3 is an example of the very rare type of congenital epiphrenic diverticulum (Janes, 1946). Granet in 1933 could only find 49 cases of epiphrenic diverticula, acquired and congenital, in the world literature, of which on analysis he accepted 31. It is, however, not unlikely, as Barrett (1933) suggested, that most of these diverticula are initially caused by extramural traction, and subsequently

attain their full dimensions through the effects of an increase in intra-œsophageal tension. In our case the congenital nature of the diverticulum is proved by the presence of pancreatic tissue in the wall. The association with bronchiectasis here, as in the case of accessory lung, is of interest. The histological findings suggestive of a congenital malformation of the bronchial tree are the same as in *Case* 1, but in both the picture is grossly obscured by the superimposed infection and there is good evidence of ulceration and destruction of bronchial walls.

The morphogenesis of this diverticulum may be discussed together with the ciliated epithelial cyst of *Case* 5.

Œsophageal cysts are unusual malformations. They have been discussed by Smith and Stone (1924), Black and Benjamin (1936), Brass (1936), Nicholls (1942), Ladd and Scott (1944), and others. They may be lined by œsophageal, intestinal, or gastric mucosa, or, rarer, by ciliated epithelium. The latter is not a surprising finding and does not suggest a derivation from the tracheobronchial tree. In an embryo of 65 mm. crown-rump length (eleventh week) the whole œsophagus is lined by columnar ciliated epithelium and even in the sixth month a few ciliated epithelial cells may be found lining the œsophageal lumen (Patten, 1947).

The explanation of the pathomorphogenesis of congenital œsophageal cysts as ' excess formations ' due to supernumerary buds of the foregut or displaced epithelial islands is accepted by the majority of authors. Ladd and Scott, however, attempted recently a different explanation. They refer to the physiological obliteration of the gut, described by Tandler. This occurs in the second month of embryonic life and is due to epithelial proliferation. Vacuolization and disintegration of the epithelium leads to the secondary formation of a lumen. Ladd and Scott assume that a disintegration in the longitudinal diameter will lead to the formation of a normal lumen, whereas a similar process in a transverse direction may cause the separation of an epithelial cyst. Their argument against a derivation of œsophageal cysts from fœtal diverticula is that the latter are most common in the region of the duodenum while alimentary duplications are found most frequently in the lower jejunum and ileum (Pachman, 1939; Ladd and Gross, 1947; McLanahan and Stone, 1934; Poucher and Mills, 1933). The same argument could be used, however, against the theory of Ladd and Scott. Patten says about the physiological embryonic obliteration of the gut: " This is particularly likely to be marked in the œsophagus and in the upper part of the small intestine, where, regularly at this age, the lumen will be found occluded by masses of epithelial cells." Although cyst formation, the result of an irregular resorption of epithelial masses, is a conceivable mechanism, the derivation from accessory buds of the foregut is a more reasonable assumption. The former hypothesis cannot be applied to such cyst formations which take their origin in the fourth to fifth week of embryonic life, nor to those which are derived from the respiratory primordium after the latter had separated from the foregut. (Cases described by Gold, 1921.)

With regard to the teratogenetic determination period it is reasonable to assume for the œsophageal diverticulum the end of the fourth week and for the ciliated epithelial cyst a later period. Such an assumption would not only do justice to the greater potentialities of the tissue in *Case* 2, evidenced by the presence of pancreatic tissue, but the shortness of the œsophagus in the fourth week followed by a rapid intercalated growth would account for the supernumerary bud being carried down almost to the extreme caudal end of the œsophagus. A point of particular interest is that the diverticulum was lined by stratified squamous epithelium while an isolated group of endodermal epithelial cells differentiated into pancreatic tissue and that the separated cyst in *Case* 4 had a lining of ciliated epithelium. Where the continuity was maintained the epithelium was under the normal influence of evocators and ' eidogens '. Where the separation was complete the dependent differentiation became substituted by self-differentiation. This resulted in arrest at the ciliated epithelial stage in one case and in a differentiation into pancreatic tissue in the other. As Foulds (1940) states in his histological analysis of tumours: " The prospective potentiality of a given part is much wider than its prospective significance."

SUMMARY

Five cases of intrathoracic malformations are described. The first was a left superior accessory lung completely separated from the bronchial tree, the second a right ' tracheal ' accessory lung, the third an œsophageal epiphrenic diverticulum with pancreatic tissue attached to its wall, the fourth an intrapericardial ' teratoid tumour ', the fifth a ciliated epithelial œsophageal cyst. An attempt is made to explain all five on the basis of an interference with organizer centres leading to a failure of resorption of accessory buds of the primitive foregut and the displacement of the growing buds within intersections of morphogenetic gradients resulting in a preponderance of self-differentiation over dependent differentiation and thus to a variety of prospective potentialities becoming manifest.

The association of an abnormal artery of the elastic type with duplications of the foregut is of practical importance. This was, however, found in only one case of the present series.

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RHABDOMYOSARCOMA OF SKELETAL MUSCLE

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THE rarity of undoubted rhabdomyosarcomas of adult voluntary muscles is particularly noteworthy (Willis, 1948). Stout (1946), presented as rhabdomyosarcomata of skeletal muscles those tumours which were definitely made up of cells which in whole or in part had some of the characteristics of rhabdomyoblasts and which had arisen in, or immediately adjacent to, striated muscles (exclusive of heart muscle). He tabulated all the published cases which he could find, and added 14 previously unpublished ones of his own. The total was 121.

Two cases have been treated in King's College Hospital during the last two years, and are worthy of being recorded.

CASE REPORTS

Case 1.—

HISTORY.—A woman aged 72 was admitted on July 9, 1947. She complained of a swelling of her right calf.

In November, 1946, she had noticed that her right foot occasionally became numb. Three months later her right ankle swelled. At the same time she noticed a hard lump, the size of a walnut, in the middle of the calf of the same leg. Cramps developed in the whole leg, shooting pains occurred in the foot, and the leg began to feel hot, especially around the calf. About three weeks after this she noticed another small mass in the right calf, very near the first lump. The cramp and shooting pains became more severe and more frequent, and pain now radiated up to the right buttock. The leg felt very hot and tender, and it became swollen as high up as the knee. She could bear nothing to touch the knee, and, though she could still walk, the pain and swelling were so much aggravated that she did as little walking as possible.

In May, 1947, the two masses in the calf coalesced, and the calf became more swollen and tense. The cramps and pain were now a little less severe, and the swelling was controlled to some extent by elevation of the limb.

During June, 1947, the cramps, swelling, and hot feeling were constant, but the calf gradually grew larger. For the last two weeks before admission the pain was, if anything, a little less severe, and the leg felt dead and heavy.

Since May, 1947, the patient had felt weak, and since the onset of the trouble in her leg she had lost considerable weight. She has suffered from chronic gastritis and chronic constipation. Otherwise she made no complaint of her general condition.

ON EXAMINATION.—The right calf was much larger than the left; at a level 3 in. below the tibial tubercle the right calf measured 17½ in., the left calf 12½ in.

There was erythema of the right leg below the knee, an area of intense redness 1 in. above and lateral to the medial malleolus, and three reddened areas on the posterior aspect of the right calf. The skin of the leg below the right knee was dry, shiny, and desquamating, especially anteriorly; there was increased pigmentation.

Œdema was present, being especially marked at the ankle.

On palpation, the right calf was very tense and tender. A mass was palpable, the size of an orange, extending from the lowest part of the popliteal fossa to a point half way down the leg. It appeared to be in the site of the medial part of the gastrocnemius muscle. The mass was not attached to skin and it moved with the

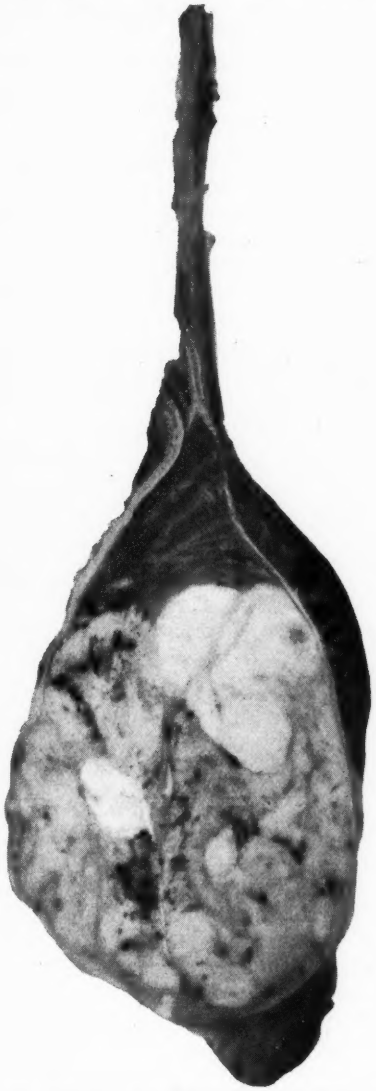

FIG. 301.—*Case* 1. Longitudinal section of gastrocnemius muscle showing a rhabdomyosarcoma in the medial head. The tumour is non-encapsulated, and small outlying nodules of growth can be seen.

gastrocnemius when the latter was contracted or relaxed. It was hard in consistency and showed no evidence of fluctuation. A well-defined edge could only be felt on the medial side of the mass.

No lymph-glands were palpable in the popliteal fossa, but the right inguinal lymph-glands were firmer and slightly larger than those on the left.

No vascular or neurological changes were found in the right leg or foot.

General examination revealed no relevant abnormalities.

Wassermann reaction was negative.

Radiographs of the right leg showed a well-marked soft-tissue swelling, but there was no evidence of bony abnormality.

TREATMENT AND PROGRESS.—On July 11, 1947, a tourniquet was applied to the upper thigh, and an incision was made into the swelling. The tissue of the swelling had a sarcomatous appearance, and an amputation through the mid-thigh was performed. Lateral flaps were used, and the stump was closed with drainage.

Convalescence was complicated by multiple pulmonary emboli, but the patient was discharged well, with the stump firmly healed, on Aug. 25, 1947.

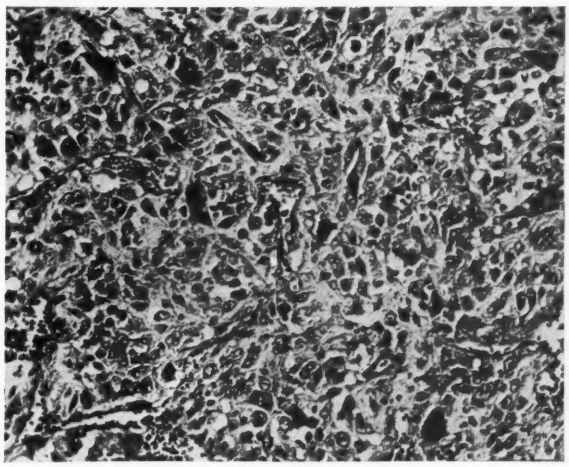

FIG. 302.—*Case* 1. Section of the rhabdomyosarcoma. Giant cells are present, and pleomorphism of the growth is clear. H. and E. (× 105.)

FIG. 303.—*Case* 1. A high-power view of a section of tumour stained with Heidenhain's iron hæmatoxylin. Longitudinal striation in the centre of the field. (× 750.)

A radiograph of the chest taken on July 24 showed no secondary deposits in the lungs.

The patient was last seen in November, 1948. She was in good health, with no evidence of recurrence or metastasis

PATHOLOGICAL REPORT (Professor H. A. Magnus).—The specimen consists of a right leg removed by an above-knee amputation. The calf is expanded by a globular tumour, into which an incision had been made at operation prior to amputation. The tumour involves only the medial head of gastrocnemius muscle, and the whole of the latter was dissected out quite easily. The tumour and muscle were sliced longitudinally (*Fig.* 301).

The growth is rounded, measuring about 10 cm. in diameter, and its cut surface is soft and fleshy. In

colour it is pinkish, with a softer area in the upper part which is yellowish-red and may be necrotic. Most of the tumour appears to be encapsulated, but there are several small nodules lying in the muscle, close to, but separate from, the main mass. No lymph-nodes were found in the popliteal fat.

Sections show a highly cellular tumour composed for the most part of fusiform cells, amongst which are scattered large irregularly-shaped giant cells. The cytoplasm of both types of cell is acidophilic and the nuclei are very large and vesicular. Some of the giant cells are multinucleate. There are quite numerous mitotic figures (*Fig.* 302).

With Heidenhain's iron-hæmatoxylin stain longitudinal and cross-striations can be seen in some of the tumour cells (*Fig.* 303), and in one section clear transition can be seen at the periphery of the tumour from normal muscle to neoplastic tissue.

The tumour is not unduly vascular. It contains only a very small quantity of fibrous tissue as stroma, and hardly any reticulum is present.

Conclusion: This tumour is a rhabdomyosarcoma.

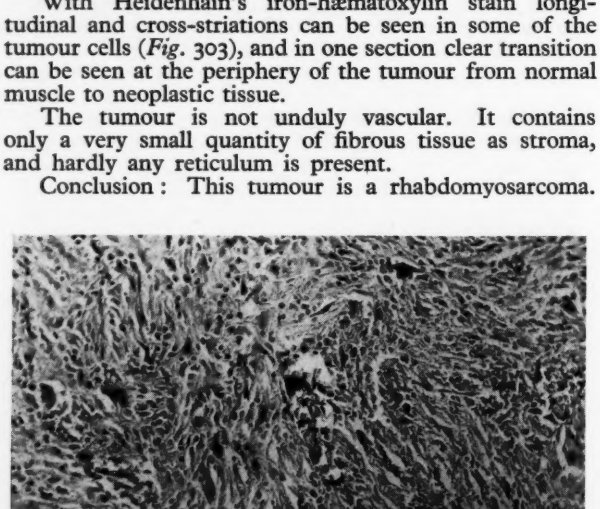

FIG. 304.—*Case* 2. A section of the rhabdomyosarcoma. The pleomorphic structure of the growth is shown, and giant cells are well in evidence. H. and E. (× 140.)

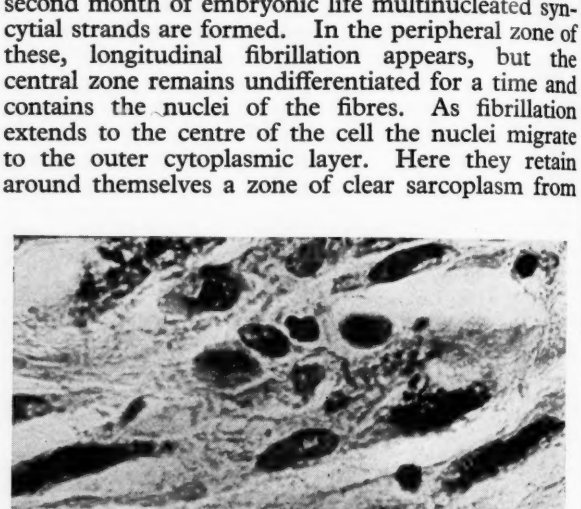

FIG. 305.—*Case* 2. A section of the tumour stained with Heidenhain's iron hæmatoxylin. Several of the cells in this field show well-marked transverse striations. (× 750.)

Case 2.—This patient, a woman aged 62, underwent a right local mastectomy on March 13, 1946. A cyst, the size of a cricket-ball and containing clear yellow fluid attached to skin and deep structures, was removed with the breast. (No pathological report on the breast is available.)

In April, 1948, a swelling was noticed underneath the centre of the scar. It measured 1 × ¾ in., and was oval in shape. It was freely mobile, being unattached deeply or to skin. There were no enlarged glands in either axilla or supraclavicular region.

General examination revealed no abnormality.

On May 27 the mass was excised widely, with the overlying skin. On June 4, the patient was discharged, healed and well.

She was last seen on Sept 14, when she was well, with no evidence of recurrence.

PATHOLOGICAL REPORT (Professor H. A. Magnus).—The specimen consists of an irregular mass of firm tissue, loosely attached to an elliptical piece of skin and surrounded by adipose tissue. The mass measures 3 × 2 × 1 cm.

Sections show the tumour to have all the histological characteristics of a rhabdomyosarcoma, as described in the report on the specimen from *Case* 1. Its origin from a small plaque of normal muscle included in the specimen removed can be clearly seen. (*Figs.* 304, 305.)

THE LITERATURE

Cappell and Montgomery (1937) described the development of striped muscles as follows: They arise, in embryonic life, chiefly from the myotomes which differentiate at an early age from the somites of the paraxial mesoblast. At first the primitive muscle cells are round and oval (the myoblasts). They rapidly become spindle-shaped, but for a time retain their mononuclear character. The spindle cells then become elongated and their nuclei undergo division without division of the cytoplasm. By the second month of embryonic life multinucleated syncytial strands are formed. In the peripheral zone of these, longitudinal fibrillation appears, but the central zone remains undifferentiated for a time and contains the nuclei of the fibres. As fibrillation extends to the centre of the cell the nuclei migrate to the outer cytoplasmic layer. Here they retain around themselves a zone of clear sarcoplasm from which the sarcolemma is derived at a later date. Transverse striation appears only some time after fibrillation is well advanced.

Thus, as it grows and differentiates into adult form, the myoblast passes through stages from a primitive round cell, through a spindle-cell form, to the multinucleate stage of the syncytial muscle-fibre. In the latter, myofibrils produce the characteristic longitudinal and transverse striation.

This recapitulation of the development of striated muscle is valuable as a guide to the understanding of the various tumours which have been described in the literature as arising in this tissue.

Abrikosoff, in 1926, first stressed the importance of the cells which he called myoblasts (Cappell and Montgomery, 1937). These round, oval, or mononuclear cells, 20–25 μ in length, had strongly acidophilic, granular cytoplasm. They were found arranged in strands or bundles; they might show longitudinal or cross-striation or they might be entirely devoid of any trace of such differentiation. According to the character of their cells, Abrikosoff distinguished four varieties of striated muscle tumour. Cappell and Montgomery simplified this

classification and described only two groups: (1) Tumours which showed well-defined cross-striation; (2) Tumours in which cross-striation was lacking.

The tumours of the first group, which were composed of well-differentiated muscle-fibres and which were devoid of primitive and anaplastic forms, Cappell and Montgomery called 'simple rhabdomyomata'. The tumours which were composed of pleomorphic round or spindle cells which show well-defined cross-striation in at least a proportion of the cells, they called 'malignant rhabdomyoma sarcomatodes'.

The tumours of the second group consisted of cells whose staining reactions were those of muscle tissue. Morphologically they resembled myoblasts of spindle shape. These cells were either devoid of striation or showed very delicate striation only by special technique, e.g., careful differentiation with Heidenhain's iron hæmatoxylin (*Figs.* 303, 305). These growths were not highly pleomorphic, and despite the absence of striation their cells were not very primitive. They showed little active mitosis. Cappell and Montgomery called these tumours simply myoblastomata.

Despite their classification, Cappell and Montgomery add that there is no absolute line of demarcation between rhabdomyoma and myoblastoma. Transitions between the two forms occur, and lead to difficulty in classification.

The origin of the granular-cell myoblastoma is not yet determined (Powell, 1946). Powell says that opinion seems to lean to Klinge's dysontogenetic theory, which states that tumours of this group arise from retained or misplaced primitive myoblasts which fail to develop into adult muscle tissue. Abrikosoff thought that the tumours arose from primitive muscle cells which developed in an attempt to repair a degenerative process following injury or inflammation; he did not think they arose from embryonal cell rests. Stout (1947) states that the origin of these tumours is generally supposed to be from a highly specialized form of myoblast, but that these cells do not produce definite cross or longitudinal striation, either *in vivo* or *in vitro*.

Willis (1948) has reached his own conclusions regarding myoblastomas. The benign granular-cell myoblastomas of the tongue and of other skeletal muscle tissue, he says, are not tumours but degenerative or regenerative lesions of the muscle-fibres. If, however, the occasional existence of true neoplasms of this appearance were to be proved, they should be called 'rhabdomyomas'. Lesions of similar appearance in tissues where no striated muscle is present should not be called 'myoblastomas'. Their muscular nature is not proved. Finally, Willis says that the term 'myoblastoma' is unnecessary and should not be applied to malignant tumours of proved muscular origin; such tumours are rhabdomyosarcomas. If anaplasia deprives the cells of a rhabdomyosarcoma of the distinctive characters of striated muscle cells, especially cross-striations, then certain histological recognition of the true nature of the tumour becomes impossible.

We can summarize the position:—

1. Benign granular-cell myoblastoma is thought by some to be a neoplasm, by others to be a reaction to injury or inflammation. No striation can be observed in the cells of this structure. The cells are large, with a granular, acidophilic cytoplasm, and they are packed together in solid masses without connective-tissue fibres between the individual cells (Stout, 1947).

2. Rhabdomyoma is a true tumour of striated muscle in which cross-striation can be demonstrated.

3. Rhabdomyosarcoma is a malignant tumour of striated muscle, including the form called by some 'malignant-cell myoblastoma'.

DISCUSSION

Willis (1948), emphasizes the youth of all the cases of undoubted rhabdomyosarcoma. Both the cases which I have reported are in elderly women, and MacCallum (1933) said that no age was exempt. Stout (1946) found that most cases occurred between the ages of 40 and 60.

No aetiological factor is determinable in either of my cases, but *Case* 2 is interesting as the tumour arose in pectoral muscle which had undoubtedly been traumatized in the performance of a local mastectomy.

The site of the tumours reported here is not unusual. Stout (1946) found that the thigh and leg were the commonest sites, and both MacCallum (1933) and Stout (1946) reported tumours arising in similar form to that of *Case* 1.

The common symptoms and signs of these tumours are by no means diagnostic. Pain and the presence of a tender swelling were present in *Case* 1. In this case the symptoms attributable to pressure from a tense swelling in the calf are of interest.

There was no evidence in either of these cases of spread to the lymphatic glands or by the blood-stream. Stout (1946) found that spread occurred by both these routes. Local invasiveness is a feature of these tumours, however, and is well illustrated in *Case* 1. It is also interesting to note in this case the outlying nodules of growth, which are separate from the main tumour mass. Such nodules may well be missed if an attempt at treatment by local excision is made (*Fig.* 301).

Treatment.—Adequate and radical removal of the tumour is the ideal treatment. All outlying nodules, such as can be seen in *Case* 1, must be removed. The literature abounds in records of cases of local recurrence. In the case of an extremity, amputation may or may not be necessary; extent of growth will decide. Radiotherapy does not appear to have been given an extensive trial in the treatment of these tumours, but such results as are reported are not favourable.

SUMMARY

1. Two cases of rhabdomyosarcoma, one involving the gastrocnemius muscle, the other involving pectoralis major, are described.

2. The literature concerning tumours arising in skeletal muscle is reviewed.

3. The clinical features and treatment of rhabdomyosarcomata are considered.

I should like to thank Sir Cecil Wakeley for permission to publish these cases and Professor

Magnus for his help with the histology. My thanks are also due to Mr. Smith, of the Photographic Department, King's College Hospital.

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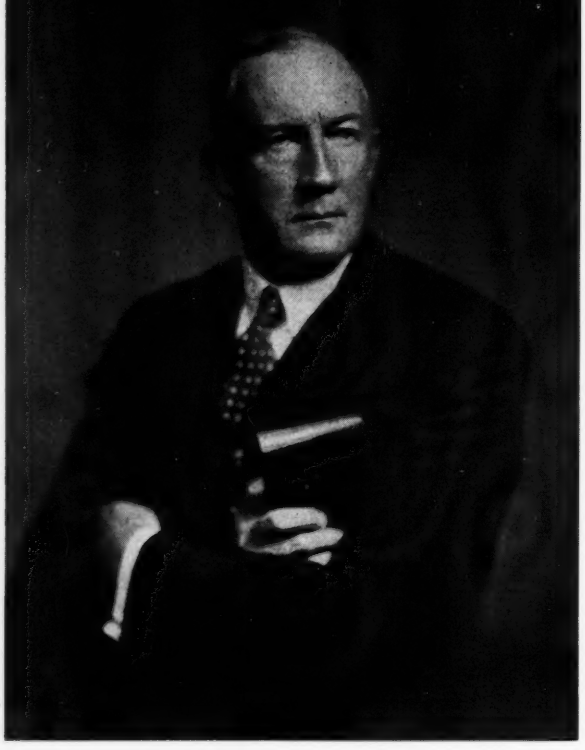
In Memoriam

SIR ALAN NEWTON

(1887—1949)

THE death of Sir Alan Newton in Melbourne on Aug. 3, 1949, robbed the southern hemisphere of its dominant surgical figure. Hibbert Alan Stephen Newton was born on April 30, 1887, and seemed from the very first to have been created in the heroic mould. Fortunate in his parentage and forbears and in the educational background of Haileybury College, Melbourne, and the University of his native city, he was destined to take the leading place, assured and unquestioned, in whatever profession or calling he elected to embrace. His particular vintage year of medical undergraduates at Melbourne University was distinguished by its brilliance, yet in this galaxy of able young men Alan Newton secured each session the premier place with ever-recurring, almost mathematical, certainty. He was elected to the Honorary Surgical Staff of the Royal Melbourne Hospital at the early age of 26.



SIR ALAN NEWTON
(1887—1949)

Alan Newton learned much of the art of surgery from his old chief, Fred Bird, but a visit to England in his early post-graduate period brought him into close contact with such stimulating personalities as Arthur Hurst and Victor Horsley; indeed, the atmosphere of Horsley's experimental neurosurgical laboratory remained an unfading influence with him, but he was also to come under the spell of Harvey Cushing in the Ypres salient in the autumn of 1917.

Although some of his early papers dealt with traumatic lesions of the central nervous system, the catholicity of his surgical interests may be gauged by reference to his writings, although his name will always be pre-eminently associated with thyroid surgery—*nil tetigit quod non ornavit*.

Nature had endowed Alan Newton with the gift of stature and with a sure inevitability he commanded notice in the crowd and in the councils and assemblies of men. None who have attained the dignity of the woolsack of the Royal Australasian College of Surgeons can have worn its presidential robe more correctly or fittingly: the imposing majesty of his appearance, the Olympian calm displayed in his carriage and mien, the decorum with which he conducted meetings or controlled examinations, befitted the dignity of his high office.

Possibly, however, he may have even achieved more in his efforts to promote the prestige and influence of the College, which he had so much at heart during the many years when he was censor-in-chief and the real "power behind the throne" of Surgical Australasia; and when the hammer-blow of fate struck home he had lived to see the realization of his dream that the Australasian "primary fellowship" should be officially recognized by the English

College of Surgeons and to hear that a Melbourne Professor of Anatomy had participated in the conduct of an English "primary fellowship" examination in London.

Those who have listened to Alan's oratory will remember his addresses for all time; two special gems were his Stawell Oration and an address to his colleagues and friends at the Royal Melbourne Hospital dinner on "The Spirit of the Place". The mastery of phrase, the wondrous imagery of words, the impeccable English, the entire absence of local accent, and the sentences that flowed with an almost contemptuous ease, commanding attention and sweeping away opposition and adverse criticism like a river in spate, place him in a class to which Moynihan or Paget belonged.

Alan Newton's cerebral fibre was such that he might have readily and ably assumed the duties of a Governor-General, Lord Chief Justice, or Chief Director of any great banking institute: indeed his reputation for probity, sound judgement, and business acumen, and his wide knowledge of men and affairs placed him on the board of directors of several of the leading companies of Australia. No account of Alan would be complete without reference to his chairmanship from 1940 to 1945 of the Medical Equipment Control Committee, Department of Defence, and in this capacity he made a major contribution to the Australian war effort; the utmost confidence was reposed in his judgement and decision by the Australian Government.

He had all the qualities needed for greatness and leadership, including the supreme one, the Old Roman *gravitas* or character. But Alan Newton was more than a leader; he was a statesman indeed. Yet this was no leaden superman, no heavy-handed egotist, but one adorned with all the charms and graces, a paladin of courtesy. His bonhomie was not born of boisterous spirits or raucous laughter; his quiet and engaging chuckle captivated his companions and friends.

The immensity of his surgical reputation brought him in contact with every rank of society: he "walked with Kings, nor lost the common touch". All knew his innate sympathy and humanity, and as he wrote in one of his addresses "the secret of the care of the sick is to care for the sick"; to this end he devoted his life, unsparing of himself.

The last years of his life were clouded with suffering and enforced retirement from active participation in his work. Perhaps, in the night watches he may have silently reflected on all that he had achieved for the advancement of Australasian surgery, and, conscious of the respect and admiration of his fellow surgeons, the adoration of his juniors, the gratitude of his patients, he may have thought, even if he did not write, "*Non omnis moriar*". Perhaps those words that Tacitus employs at the close of the *Agricola* proclaim Alan Newton's niche in the shrine of Australian surgery: *Quidquid amavimus, quidquid mirati sumus, manet mansurumque est in animis hominum in aeternitate temporum*; . . . *posteritati narratus et traditus superstes eris.**

G. G–T.
C. L. R.

* What we loved, what we admired in you endures and will endure in the souls of men through the endless course of the ages. Your name and fame will survive in the minds of posterity.

SHORT NOTES OF RARE OR OBSCURE CASES

LEIOMYOMA OF TERMINAL ILEUM

By JOHN BORRIE

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LEIOMYOMATA of the small intestine are uncommon, and the majority of them warrant publishing so that an idea of their frequency and varying characteristics may be ascertained from the literature.

CASE REPORT

Mrs. D. R., aged 45, was admitted on Jan. 16, 1947, for investigation of recurrent attacks of melæna since 1931. During the past sixteen years she had nine severe hæmorrhages, occurring in 1931, 1935, 1937, and 1939, when she was incapacitated for a period of twelve weeks on each occasion. There were also three hæmorrhages in 1944 and two in 1946. The attacks of melæna were heralded by giddiness and shortness of breath, followed by peri-umbilical pain, which persisted for 24 hours after bleeding. There was never any hæmatemesis. At no time had she definite symptoms referable to the œsophagus, stomach, or duodenum, nor did X-ray examination of these organs reveal any abnormality. She felt weak, had a poor appetite, and weighed only 6 st. 6 lb. In the preceding six weeks she had lost 4 lb. in weight. The past history and family history were essentially negative. She was nulliparous.

The temperature and pulse-rate were normal, and the blood-pressure 120/80 mm. Hg. Except for three primary pile masses, the alimentary track was normal. Rectally no mass was detected, and examination of the other systems revealed no abnormality. The hæmoglobin was 87 per cent, and the blood-urea 34 mg. 100 c.c. Although no definite pre-operative diagnosis was made, duodenal ulceration was considered. Because of the seriousness of the hæmorrhages, however, and the chronic ill health they caused, laparotomy was advised.

AT OPERATION.—On Jan. 18, under pentothal, gas, oxygen, and cyclopropane anæsthesia, the abdomen was explored through a midline upper abdominal incision. The stomach, duodenum, and gall-bladder were normal. Arising from the vascular arch formed by the anastomosing right and left gastro-epiploic vessels was a large aberrant artery with its accompanying vein (*Fig.* 306). It passed downwards along the left border of the great omentum, ending as a free vascular cord which lay anterior to all coils of small intestine. This vascular cord disappeared from

view over the pelvic brim. Palpation revealed that it ended in an irregular mass which was adherent both to the floor of the true pelvis and to a loop of small intestine. A small intramural fibromyoma on the posterior surface of the uterus was also observed.

Because of these findings the upper abdominal wound was closed and a left infra-umbilical paramedian incision made.

Procedure.—The aberrant artery and vein were divided at the level of the pelvic brim, just below their junction with the great omentum. The irregular pelvic mass, bright red in colour, was separated from the walls of the pouch of Douglas by blunt dissection, and delivered into the wound. The raw pelvic surface was covered with a hot saline pack. At that time the mass was thought to

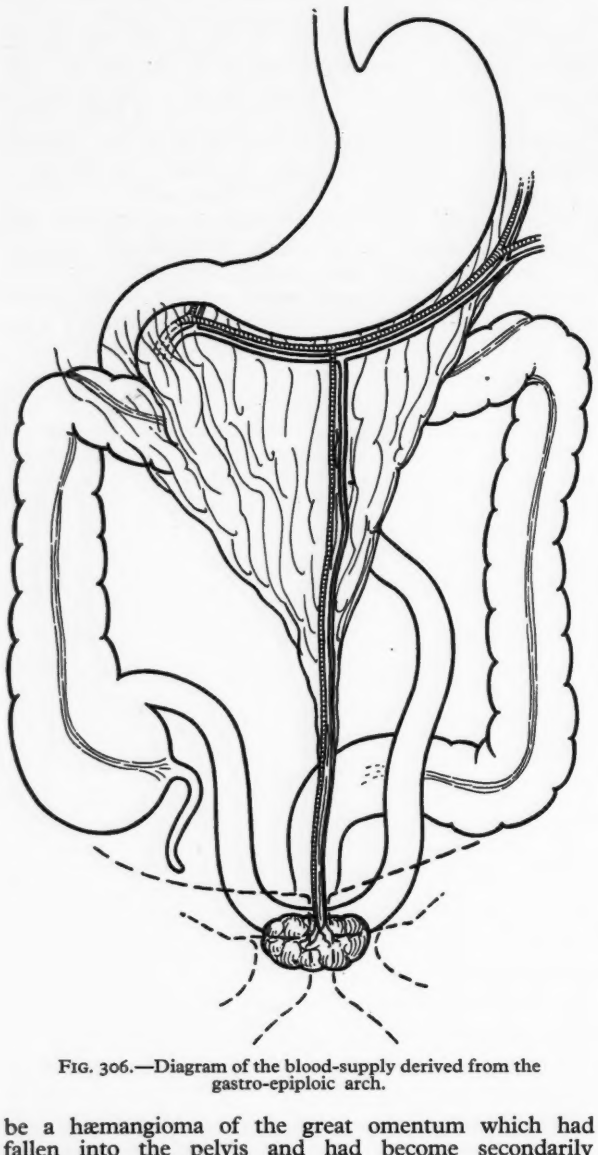

FIG. 306.—Diagram of the blood-supply derived from the gastro-epiploic arch.

be a hæmangioma of the great omentum which had fallen into the pelvis and had become secondarily adherent to the last loop of the small intestine, tethering it firmly down.

The tumour, which was attached only in one small area to the antimesenteric border of the small intestine, was excised by a V-shaped cut, bowel continuity being restored by transversly placed mersutures. The wound was closed with catgut for muscle layers, and deknatel for the skin. During the operation one pint of blood was given.

POST-OPERATIVE COURSE.—A Ryle's tube, left in the stomach, was aspirated hourly.

Jan. 19: There was some nausea, but no abdominal distension; fluids were taken by mouth.

Jan. 20: The abdomen was soft, borborygmi heard, but no flatus passed. The Ryle's tube was removed in the evening. Temperature 100° F.

Jan. 21: As the evening temperature was 101° F., a course of penicillin, 50,000 units 3-hourly for three days, was started.

Jan. 22: The patient passed flatus three times in the night.

Jan. 23: Following a glycerin suppository, the bowels opened.

Jan. 24: Sutures removed and wound healthy.

Jan. 31: Wound now well healed, bowels opening daily.

Feb. 4: Discharged.

On Sept. 30, 1948, she wrote that she felt better and stronger than since 1931, that her weight was steady, her bowels regular, her wound healed, and that there had been no bleeding since operation.

PATHOLOGY.—The pathological report was as follows:—

Gross Description.—Piece of omentum with a lobulated tumour 4 × 2½ in., arising from the free border. There is an elliptical piece of small intestine resected from the antimesenteric border. The cut surface shows a solid mass of pale new growth with hæmorrhagic areas.

Microscopy.—Benign leiomyoma, partly well preserved, partly engorged and hæmorrhagic. (The tumour could have arisen from either the small intestine itself or from the uterus, with secondary attachment to the omentum and intestine.) (*Figs.* 307, 308.)

DISCUSSION

The site of this tumour and the unusual source of its blood-supply is striking. I surmise that it grew primarily in the terminal ileum, and had prolapsed secondarily into the pelvis, dragging its parent ileal loop, besides some adherent omentum, with it. In time the omental strand had become attenuated, leaving a strap-like artery and vein crossing the abdomen, ventral to all coils of small intestine. As the tumour grew larger, so its secondary omental blood-supply increased, with corresponding increase in size of the right and left gastro-epiploic vessels.

Although a small uterine fibromyoma was felt at operation, because the uterus was quite free and devoid of all adhesions, there was nothing to suggest that the ileal tumour was of uterine origin.

Gordon-Taylor (1943) has emphasized the importance of remembering leiomyoma of the intestine in patients frequently admitted to hospital for unexplained high-intestine bleeding; and he records two such examples where one ended fatally after laparotomy for severe melæna. In reviewing past literature he mentions that, rarely, a palpable tumour, or some bowel complication such as intestinal obstruction or intussusception, may reveal the cause of this bleeding. It may be severe enough to cause death. In the belief that it had come from a silent duodenal ulcer, unnecessary gastrojejunostomy has even been performed. Often careful laparotomy alone can reveal the truth.

After discussing recorded incidence, pathology, and clinical characteristics, Hanno and Mensch (1944) described a patient aged 53 whose history dated from 1930. By 1933 he had had five attacks of melæna, after which gastro-enterostomy was

performed. In the next eleven years there were fifteen further hæmorrhages, of which the last was fatal. Thirteen months was the longest time between any attack. My patient had had nine hæmorrhages in sixteen years, the longest interval between any two being five years.

FIG. 307.—Photomicrograph of leiomyoma of terminal ileum. (Low-power magnification.)

FIG. 308.—Photomicrograph of leiomyoma of terminal ileum. (High-power magnification.)

Steiner (1898) divided myomata into submucosal and subserosal varieties. According to Smith (1937) submucosal myomata tend to produce intestinal obstruction by intussusception, hæmorrhage by superficial ulceration, or may occasionally give rise to ulcer-like pain. It is the subserosal type which grows the larger, and undergoes focal hæmorrhagic necrosis. These necrotic areas tend to merge and rupture through the tumour tissue into the intestinal lumen, with unexpected dangerous hæmorrhages from the shaggy centre. This centre may slough away completely, leaving a diverticulum. Such a case was reported by Allen and Sniffen (1945), where a leiomyoma of the terminal ileum contained a diverticulum, 7 mm. in diameter, that extended 1 cm. into the substance of the tumour.

Because a similar diverticulum became filled with barium on fluoroscopy, Jones and Brubaker (1942) considered that in their patient the large leiomyoma which had also prolapsed into the pelvis might well have arisen in a Meckel's diverticulum.

The tendency for these tumours to prolapse into the pelvis is of interest, and suggests that a careful rectal examination in patients with unexplained melæna might disclose the cause more certainly than other methods. In 1947 Cookson described a tumour that entirely filled the true pelvis, producing large-gut obstruction.

As barium is not impeded and the intestinal lumen not distorted, radiological assistance in diagnosis cannot be anticipated by any of the usual methods. Patterson and Geer (1943), however, reported excising a leiomyoma where previous fluoroscopy had shown a stenosing lesion of the small intestine in a patient with melæna as the leading symptom.

SUMMARY AND CONCLUSIONS

1. A case of leiomyoma of the terminal ileum is reported.

2. The tumour derived its main blood-supply from the gastro-epiploic vascular arch.

3. ' Unexplained melæna ' suggests ' leiomyoma '.

4. Clinical and radiological investigations are usually negative.

5. Often laparotomy alone will reveal the truth.

My thanks are due to Mr. Maingot for his help with this paper; to Professor R. Willis for the pathological report and sections; and to Mr. Shepley of the Department of Surgery, Edinburgh, for his drawing of the lesion.

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PRIMARY CARCINOMA OF THE URETER

By A. L. CARRODUS and V. S. HOWARTH, Sydney, N.S.W.

Primary carcinoma of the ureter is exceedingly rare. Lazarus and Marks (1945) have fully reviewed its symptomatology and pathology and have collected 183 examples recorded in the literature, and a recent report by Stock and Wells (1948) records 2 further cases.

An additional example of a primary carcinoma of the ureter associated with a simple renal cyst warrants record.

CASE REPORT

History.—A male, aged 67, complained of intermittent hæmaturia of nine weeks' duration. He appeared quite healthy and apart from minor ailments had been well up till six months previously, when he had noticed nocturnal frequency of micturition and a diminution of the force of his urinary stream. For the past nine weeks his urine had been intermittently profusely blood-stained, with passage of clots on occasions, and once he had suffered an attack of retention of urine which was relieved following the passing of a blood-clot per urethram.

On Examination.—Clinical examination of the patient's heart, chest, and abdomen disclosed no abnormality. Cystoscopic examination revealed a trabeculated bladder and intra-urethral enlargement of both lateral lobes of the prostate gland. A long worm-like blood-clot was seen exuding from the left ureteric orifice. A plain radiograph with opaque ureteric catheters in position was taken (*Fig.* 309).

An instrumental urogram was performed (*Fig.* 310).

At Operation.—The operative approach was made through an oblique lumbo-inguinal incision. On delivery of the kidney on to the loin, a large cystic swelling was seen to occupy its convex border, and on palpation of the ureter in the vicinity of the rim of the bony pelvis, a hard spindle-shaped tumour was felt. Left nephrectomy and partial ureterectomy was performed, removal of the remaining portion of the ureter being carried out at a later date.

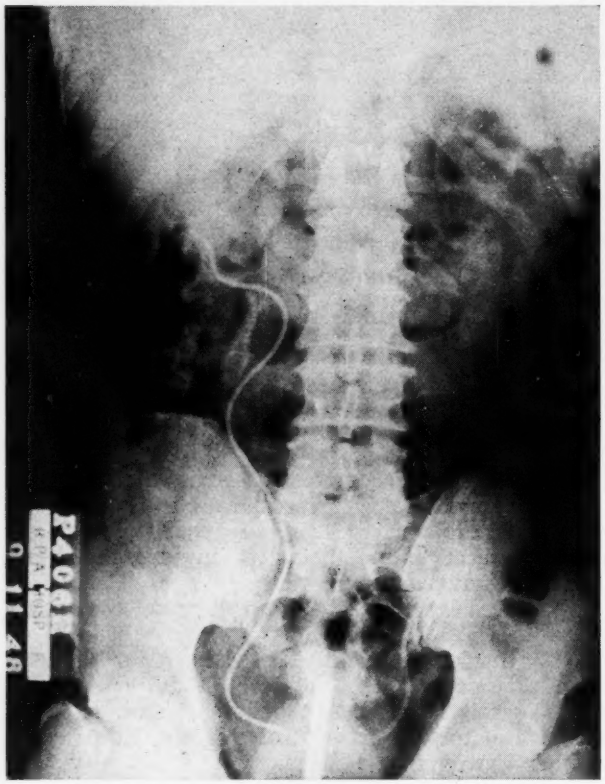

Fig. 309.—X-ray picture showing that the right catheter has taken a sinuous course to the right renal area but that the left has passed no farther than the brim of the bony pelvis. No radio-opaque calculus is to be seen in the urinary tract.

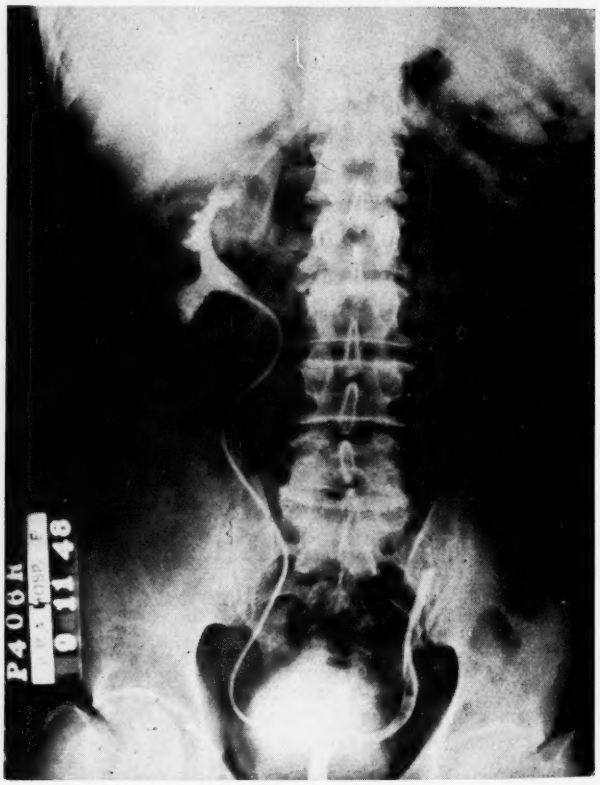

Fig. 310.—Showing a normal rena pelvis and kidney shadow on the right side, but the iodide injected into the left catheter has not penetrated past the brim of the bony pelvis and the upper border of the opaque column presents an irregular margin.

The Specimen (*Figs.* 311, 312).—Microscopical examination of the ureteral tumour showed it to be a papillary carcinoma of the transitional-cell type (*Figs.* 313, 314).

SUMMARY

1. An example of primary carcinoma of the ureter is recorded.

2. The association of the condition, perhaps fortuitous, with a simple renal cyst, clouded the operative diagnosis.

FIG. 311.—A cystic swelling is seen to arise from the convex border of the left kidney. The cyst measures 7 × 5 cm. and in the ureter, 9 cm. from the uretero-pelvic junction, there is a spindle-shaped tumour which is white in colour and measures 5 × 1·5 cm.

FIG. 312.—Section of the specimen showing the lumen of the ureter to be completely obliterated by the tumour, which has infiltrated through its wall. In a portion of it a papillary structure can be seen occupying the original lumen of the tube, but the intra- and extramural portions are solid. The ureter above the tumour is only moderately dilated and a minor degree of intrarenal hydronephrosis is present. The kidney cortex is occupied by a simple cyst which springs from the middle of the convex border.

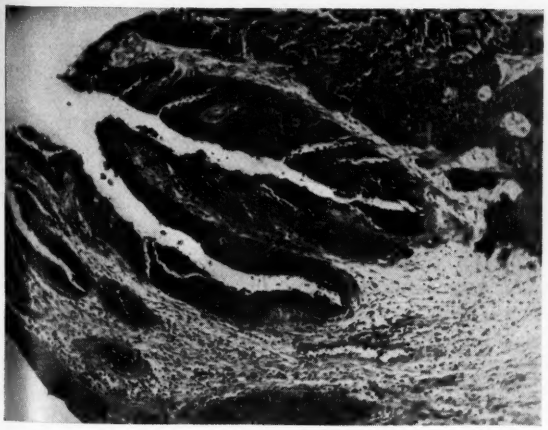

FIG. 313.—Microscopical picture of the tumour, showing it to be transitional-cell carcinoma of papilliferous type. H. & E. (× 66·6.)

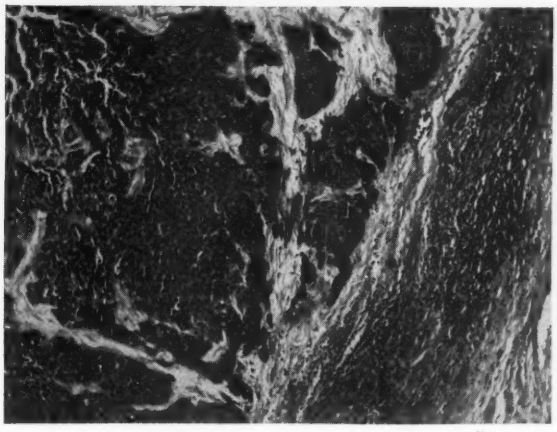

FIG. 314.—A section taken from the extramural part of the growth, showing a loss of the papillary structure, the neoplasm being formed by masses of transitional cells which have excited a marked lymphocytic tissue reaction. H. & E. (× 66·6.)

We are indebted to the Department of Medical Artistry, The University of Sydney, for the photographs.

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ADENOMATOID LEIOMYOMA OF THE EPIDIDYMIS

By W. WEATHERSTON WILSON, Wigan

Benign solid tumours of the epididymis are of rare occurrence. Thompson (1936), reporting 13 cases of solid tumours of the epididymis from the Mayo Clinic, found 60 per cent to be malignant and 40 per cent to be benign.

The proportion in a similar series published by Scalfi (1936) was 69 per cent malignant to 31 per cent benign.

Of the benign tumours leiomyoma is the most common; in 1941 Friedman and Grayzel (1942) were able to find records of only 13 cases in the literature, and since then I have been able to find accounts of 2 further cases (Halpert, 1941; Malisoff and Helpern, 1943).

A tumour of this type has never been known to recur or metastasize (Beneventi, 1947).

CASE REPORT

A 46-year-old bus driver complained of a painless lump in the left testis which he had noticed for three months. On examination, a smooth firm nodule 1·5 cm. in diameter, which was not tender, could be palpated in the lower pole of the left epididymis. It was not translucent. Nothing unusual was felt in the testis or spermatic cord, and the seminal vesicles were normal to palpation. Aortic lymph-nodes could not be felt.

Fig. 315.—Naked-eye appearance of tumour.

At operation on Oct. 7, 1948, the nodule in the epididymis was excised.

The pathologist reported: "Naked eye: an encapsulated white nodule 2 cm. in diameter. The cut surface shows the whorled arrangement of a fibromyoma. Microscopical: fibromyoma of the epididymis" (*Figs.* 315, 316).

Further examination of the sections of this tumour showed that though most of the tissue was smooth muscle (*Fig.* 317) there were areas which consisted of circular spaces lined by flattened cells (*Fig.* 318). In addition, there were groups of lymphocytes.

DISCUSSION

Tumours of this type were at one time considered to be malignant, and experienced pathologists have differed considerably in their classification of them. This is of the same type as those described by Halpert (1941) and Malisoff and Helpern (1943) as 'mixed leiomyoma and lymphangioma'. There is general agreement that the myomatous elements of these tumours represent smooth muscle, as is clearly shown when van Gieson's stain is used. There is by no means the same unanimity of opinion in regard to the cystic element. Halpert (1941) and Malisoff and Helpern (1943) believe that this tissue is lymphangiomatous, since staining of frozen sections with Sudan III does not reveal the presence of lipoids, the cystic spaces closely resembling areolar tissue in the paraffin sections. On the other hand, very similar vacuolated spaces are to be seen in some of the adenomatoid tumours of the epididymis described by Golden and Ash (1945), who state that the gland lumina contain no material staining with hæmatoxylin and eosin such as is seen in lymphangiomas. Sworn, Marshall, and Edwards (1946) suggest the term 'mixed tumour of the epididymis', but I prefer 'adenomatoid leiomyoma' because the nature of the gland-like spaces found amongst the smooth muscle-fibres has not yet been determined. No incontrovertible evidence has yet been given as to the nature of these tumours, but it seems unlikely that they are of inflammatory origin as suggested by Obendorfer (1932).

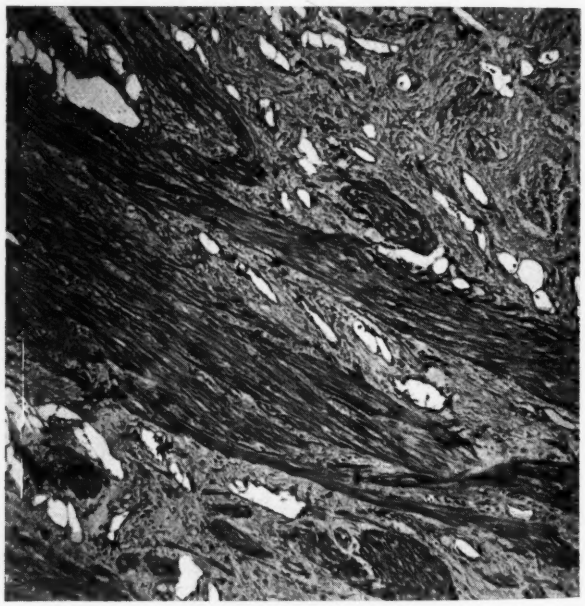

Fig. 316.—Microphotograph of tumour. (× 70.)

It is a pleasure to thank Professor S. L. Baker for his advice; Dr. R. Whitehead for constructive

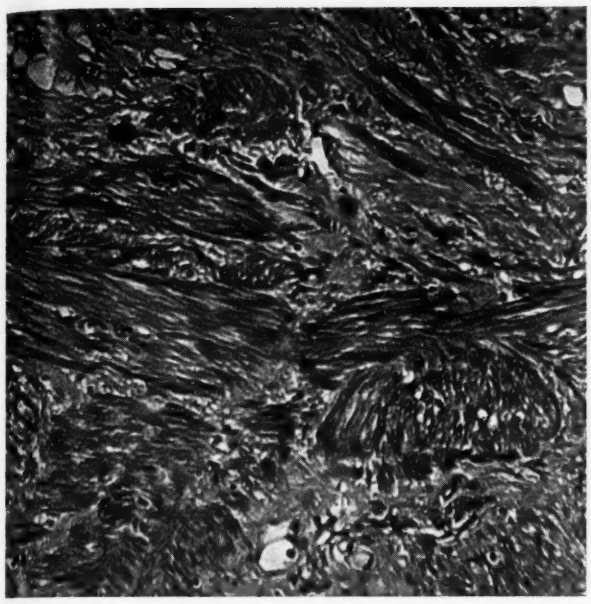

FIG. 317.—Microphotograph of tumour showing smooth muscle element. (× 140.)

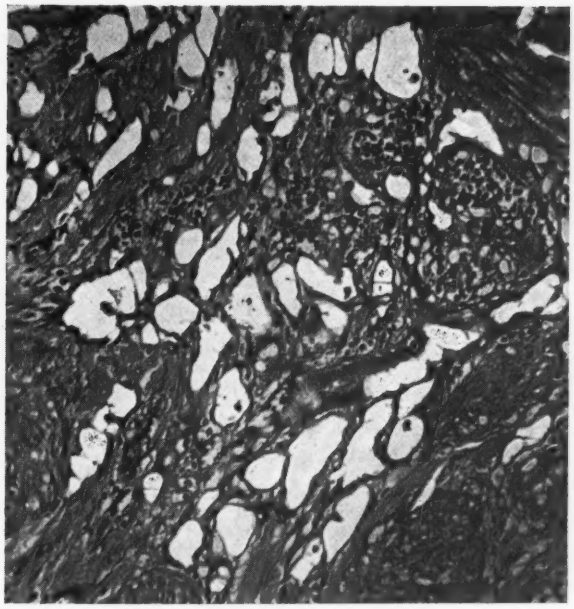

FIG. 318.—Microphotograph of tumour showing area of cystic spaces lined by flattened cells and groups of lymphocytes. (× 140.)

criticism; Mr. R. L. Holt for permission to publish this case; and Mr. Fred Ward for the microphotographs.

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A CASE OF TRAUMATIC ANEURYSM OF THE RIGHT SUPERFICIAL TEMPORAL ARTERY

BY FREDERICK SMITH
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TEMPORAL aneurysm is not very common. Winslow and Edwards, in a most excellent and complete review of the literature up to 1934, collected records of 93 cases. Of this total 79 were classified as traumatic, 9 spontaneous, and 5 cause unknown. Of the 79 traumatic cases, in 41 the aneurysm was associated with an open wound and in 33 with a contusion only.

Since their article I can find record of one further case added by Brown and Mehnert in 1942. There may be others reported from foreign sources, but I have not been able to obtain these papers.

CASE REPORT

This lesion occurred in a young man aged 34 years, whose occupation was that of steel erector.

In June, 1947, while drilling a steel girder he chanced to turn his head suddenly and struck his right temple against a projecting iron bar. The blow caused him slight pain which was momentary and he thought nothing more of the incident. No wound or bruising occurred. Some days later a small lump appeared in the right pre-auricular region. It was painless and did not increase in size, so he did not seek medical advice at that time. Indeed, he had not connected its appearance with the accident some days previously. The lump did not throb or produce any disagreeable sensations, but he noticed that he could feel it pulsate when he fingered it.

Eventually, for cosmetic reasons, he reported to his Works Medical Officer, who diagnosed the swelling as an aneurysm and sent him for surgical treatment on Jan. 12, 1948.

ON EXAMINATION.—There was a round swelling ½ in. in diameter situated in the right pre-auricular region over the line of the superficial temporal artery. The lump was superficial but unattached to skin, smoothly rounded, mobile vertically, and of a solid consistency. It was not visually pulsatile, but faint pulsations synchronous with the heart-beats were felt on palpation. Some reduction in size occurred with firm pressure. Superficial temporal arterial pulsations were palpated above and below the swelling. Obliterative pressure on the artery above or below produced no change in the swelling, but pulsations ceased when pressure was applied on both sides. No bruit heard on auscultation. No alteration

from normal appearance or texture of the skin of the head and neck. No cutaneous vascular dilatations seen. His general health was excellent. Cardiovascular and other systems normal. Blood-pressure, 124/70. Wassermann and Kahn negative.

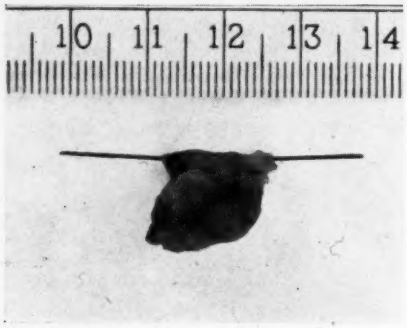

FIG. 319.—Photograph of specimen.

AT OPERATION.—A small saccular aneurysm of the right superficial temporal artery was found and treated by excision with ligation of the artery above and below (*Fig.* 319). The sac was three-quarters filled with organized clot and communicated with the artery by a pinhead-sized opening, thus explaining the solid feel, the inability to empty it completely by pressure, and the lack of systolic bruit pre-operatively (*Fig.* 320).

I would like to thank Mr. A. Elliot-Smith for his permission to publish this case, and I am also indebted to Dr. G. A. C. Summers for the photographs of the specimen.

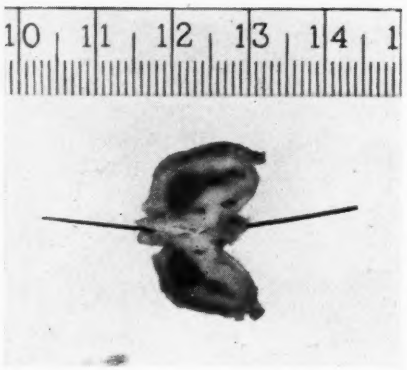

FIG. 320.—Specimen split to show interior of sac.

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HÆMOPERITONEUM FOLLOWING RUPTURE OF A GRANULOSA-CELL TUMOUR OF THE OVARY

BY J. N. WARD-McQUAID
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ONE of the rare causes of hæmoperitoneum is hæmorrhage from rupture of a granulosa-cell tumour of the ovary. These tumours are relatively rare, Hodgson, Dockerty, and Musset (1945) giving their incidence as 1·63 per cent of all ovarian tumours. The tumours are œstrogenic in action and occur most frequently in elderly women. They may then be a cause of post-menopausal bleeding. During the child-bearing years they give rise to menstrual irregularities, and in the young to precocious puberty. Acute abdominal symptoms may result from torsion of the tumour. While hæmorrhage into these tumours is also common, hæmoperitoneum from subsequent rupture of the tumour wall is extremely rare. Brewer (1948) described such a case, which presented as an abdominal emergency, and at operation over a pint of blood and clot were found in the peritoneum. He stated that he was unable to trace a similar case. A further example of severe hæmoperitoneum following rupture of a granulosa-cell tumour is now described.

CASE REPORT

HISTORY.—E. S. (R.I., No. 93026/48), aged 40, a housewife, was admitted at noon on Sept. 15, 1948, to the Radcliffe Infirmary, complaining of lower abdominal pain.

Early the previous evening she noticed a gradually increasing generalized abdominal pain. Two hours later she took a dose of salts, then fainted, and on recovering vomited three times. By midnight the pain was less, she slept on and off, and next morning the pain was in the lower abdomen. She felt sick but did not vomit. Her bowels had acted regularly, there was no urinary upset, and there was no history of recent strain or injury.

She had two similar but less severe attacks in early April and early August, 1948; each lasted two days.

Her periods began at 12 years and lasted for four days every twenty-eight days. At the age of 34, after five years of married life, she had a son. Two years later the periods became scantier and irregular, and after six months ceased altogether. During this time, the latter half of 1944, she had headaches and hot flushes. After over three years further small losses occurred, each for three days, at the end of March, in May, and at the end of July, 1948. Since 1944 she had lost a stone in weight. She had noticed no change in her breasts in recent years. There was no other relevant previous nor family history.

ON EXAMINATION.—Temperature 99·8° F. Pulse 120. Respiration 22. Blood-pressure 100/70 mm. She was slightly flushed and her tongue moist and furred. Her breath was foul and she noticed an unusual bad taste in her mouth. Her teeth were decayed. The abdomen moved on respiration, the right iliac fossa was tender and guarded, and there was release tenderness in the left lower abdomen. Bowel sounds were present. On rectal examination there was slight tenderness anteriorly. The urine was normal, and no other abnormalities were found on clinical examination.

A diagnosis of acute pelvic appendicitis was made.

OPERATION.—Under general anæsthesia a right iliac muscle-splitting incision was made, later extended

medially into the rectus sheath. The peritoneum contained free dark blood, and the pelvis was full of grumous blood-clot. After evacuating much of the blood and clot the right ovary and tube were delivered. The tube was normal, but the ovary appeared to have ruptured and consisted of blood-clot, whitish-yellow cellular material, and the ragged remains of a fibrous covering. There was no evidence of torsion. The uterus and left tube appeared normal; the left ovary was atrophic. The right tube and ovary were excised and the pedicles buried under the round ligament.

Further clots containing whitish cellular material were cleared from the pelvis, one of them lightly adherent to the pelvirectal junction by a fibrous covering which, in retrospect, must have been part of the tumour wall. This required separation by sharp dissection. In all, some 1½ pints of clot were removed, together with rather more than a pint of free blood.

The abdomen was closed without drainage. A blood transfusion, total 3 pints, was started during operation.

THE SPECIMEN (R.I.S.H. 3957/48).—The fresh tissue consisted of blood-clot with grey and white tags resembling placental tissue. Two and a half inches of apparently normal tube were loosely attached by the ovarian fimbria to a thin shell of ovarian tissue. The rest of the tissue was fragmented, and reconstruction was impossible.

HISTOLOGY.—Microscopical examination revealed a granulosa-cell tumour of predominantly diffuse structure, but the several areas examined showed wide variation. There were occasional areas of cylindromatous and microfolliculoid structure, and a few Call-Exner spaces and some larger cystic spaces were seen. The material was highly vascular, and there were areas of hyaline degeneration in the stroma. Polymorphonuclear leucocytes were abundant in the vessels and in the blood-clots. The ovarian remnant had a loose fibrous structure, and many fibrous vascular tags were attached. The tubal mucosa was hyperplastic.

PROGRESS.—There was slight vaginal bleeding for three days after operation. Apart from a low evening pyrexia of 99°–100° F., which gradually settled, convalescence was uneventful.

On Oct. 4 a gynæcological examination did not reveal any residual evidence of the granulosa-cell tumour. It was suggested that unless there was further evidence of pelvic growth, no additional surgery was indicated, and periodic observation of the patient was advised.

When seen nearly a year after operation, she was well, and there was no abnormality on examination. Her periods had recommenced, and were normal.

DISCUSSION

Rhoads (1946), in reviewing 21 of these tumours, found that no less than 5 were associated with a premature menopause, as in the reported case. Continued hyperœstrinism apparently produces amenorrhœa or hypermenorrhœa: the former may be the result of pituitary suppression. The usual finding of an enlarged uterus was not noted in this case. Slight post-operative uterine bleeding was the only symptom of œstrin withdrawal, but profuse hæmorrhage is often seen, and has even necessitated intra-uterine packing (Spencer and Hollenbeck, 1947).

The ruptured granulosa-cell tumour reported by Brewer (1948) was histologically of the diffuse type, and its wall was described as thick. In this case the tumour was predominantly of the diffuse type and the thickness of its wall, although variable, was of the order of 1 mm. There was no evidence of torsion nor history of trauma to account for the excessive bleeding in either case.

Novak (1947) refers to these tumours as 'granulosa-cell carcinomas' and in reviewing 32 cases (Novak and Brawner, 1934) states that the clinical malignancy rate was 28·1 per cent. Hodgson and others (1945), in a comprehensive survey of 62 cases, give a recurrence rate of between 6·4 and 8 per cent and analyse a formidable list of associated pathological lesions. Of their 38 post-menopausal cases, 8 developed endometrial carcinoma, and of these 3 developed carcinoma of the breast. They state that œstrogen stimulation appears to be a factor in carcinogenesis in laboratory animals, and a relation between hyperœstrinism and the development of carcinomata in the human female is implied. Seegar Jones and Te Linde (1945), stating that various authors assess the degree of malignancy from 10 to 55 per cent, illustrate the difficulties of prognosis by 3 cases, all originally considered benign, which recurred 15, 16, and 19 years after operation. In one of these the radical operation of bilateral salpingo-oöphorectomy and total hysterectomy had been performed. All 3 died within four years of recurrence.

Schulze (1933) reported a case of a granulosa-cell tumour diagnosed at operation as an ovarian pregnancy and treated by local excision. The tumour had been ruptured during removal and the contents resembled infarcted placenta. On section it was first thought to be a medullary carcinoma of the ovary, but later was found to be a folliculoid granulosa-cell tumour. The patient, who was only 27, wished for a family, and the radical operation was not performed. A son was born two years later, and the patient was alive fifteen years after operation.

Many gynæcologists perform the radical operation in all causes of ovarian neoplasms found in fit patients past the menopause. They consider it especially necessary in granulosa-cell tumours—an unduly high proportion of which are of proven malignancy—and reserve the local operation for such of these tumours as are benign on section and occur in younger women. Others believe that even past the menopause a local excision of a granulosa-cell tumour is adequate, provided it is found to be benign on section. Such tumours may, however, later prove to be clinically malignant. A possible further argument for the radical operation is that it prevents the severe bleeding which may follow a local removal. X-ray treatment has not been notably successful in the treatment of these tumours, but has been useful as a palliative measure.

The radical operation would have been performed in the case reported had it been realized that the hæmorrhage arose from an ovarian neoplasm which had ruptured. However, the grumous cellular material was thought to be of chorionic origin and the rare diagnosis of a ruptured ovarian pregnancy was made. The dissemination of tumour cells following such bleeding and disintegration of the tumour must have been considerable. It would, however, have been greater had the clot been left to absorb, as is sometimes done in a ruptured ectopic gestation. The radical operation was not performed during convalescence because of the rupture of the tumour before operation, its benign nature on section, and the clinically adequate local excision of the growth.

SUMMARY

A case of severe hæmoperitoneum following spontaneous rupture of a granulosa-cell tumour is reported, and the treatment discussed.

I am indebted to Mr. A. Elliot-Smith for permission to publish this case, and to Dr. G. A. C. Summers, of the Department of Pathology, for the histological report.

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A CASE OF PARTIAL ATRESIA OF THE PYLORIC ANTRUM DUE TO A MUCOSAL DIAPHRAGM OF DOUBTFUL ORIGIN

BY C. PATRICK SAMES

PARTIAL atresia of the stomach would appear to be excessively rare; and this is the reason for reporting the following case. After an extensive search no similar case could be found reported in the literature.

CASE REPORT

M. W., a married woman and a housewife, aged 40, has suffered for the past six years from epigastric pain associated with the taking of food. The pain was subjected to remissions, and was relieved by alkalis and sometimes by food. During the last two years she noticed that she vomited whenever she took large meals, and this vomiting usually gave some relief of pain. Nineteen months prior to admission she had a melæna, but she never had any hæmatemesis. Two years previously she had been admitted to another hospital, where she was diagnosed as having a gastric ulcer and treated on medical lines, but with little relief. Barium-meal X-ray examination showed a large gastric ulcer high up on the lesser curvature and what was later recognized to be a diaphragm-like obstruction about 1 cm. proximal to the pylorus (*Fig.* 321). There was no residue of barium after four hours.

FIG. 321.—Barium-meal radiograph showing obstructing diaphragm.

AT OPERATION (C. P. S.).—A subtotal gastrectomy was performed with removal of the gastric ulcer, pylorus, and first part of the duodenum; an antecolic gastrojejunostomy was performed to restore the continuity.

THE SPECIMEN.—Examination of the specimen showed the following macroscopical changes (*Fig.* 322):

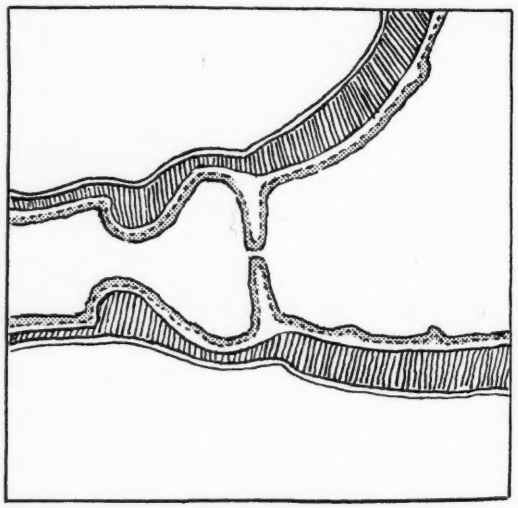

FIG. 322.—Diagrammatic representation of diaphragm.

The stomach was somewhat larger than normal and exhibited a linear depression circumferentially placed on the serosal aspect, 1·5 cm. proximal to the pylorus. There was a large gastric ulcer on the lesser curvature, and a marked muscular hypertrophy of the stomach wall stopping short at a diaphragm-like obstruction, situated 1·5 cm. proximal to the pylorus. The position of this diaphragm corresponded to the depression previously noted on the serosal surface. The diaphragm measured 3 mm. in thickness and appeared to be formed of a double layer of mucosa and submucosa. There was a small eccentric opening situated slightly nearer the greater than the lesser curvature. The stoma measured 3 mm. in diameter before being fixed and had slightly inflamed

margins. The orifice could be gradually dilated to admit a diary pencil (5 mm.). The gastric mucosa elsewhere was normal except that rugæ were rather fewer than usual. Microscopical examination of the diaphragm showed it to consist of a double layer of gastric mucosa together with the muscularis mucosæ and a substantial part of the submucosa.

DISCUSSION

The aetiology of such a condition is obscure. The first possibility that must be considered is that the diaphragm is congenital in origin. Many authors have made extensive reviews of congenital atresias of the intestinal tracts both partial and complete (Webb and Wangensteen, 1931; Sheldon, 1926; Spriggs, 1912). In all these collected reports the stomach seems peculiarly exempt as a site for such lesions, and Webb and Wangensteen collected as many as 500 cases of atresia confined to the œsophagus, duodenum, or small and large intestine. (They specifically excluded the pylorus, rectum, and anal canal.) The majority of such cases of atresia are described from post-mortem reports on stillborn children or those dying in the first few days of infancy, although there is no doubt that partial atresia can present later in life with symptoms of intestinal obstruction. Reid (1948) describes two such lesions occurring in the cæcum of a man aged 41, who had had intermittent symptoms of abdominal pain suggestive of obstruction since the age of 14.

If the diaphragm is considered to be congenital in origin the absence of obstructive symptoms prior to the age of 38 may be fortuitous but not impossible; for provided the stomach contents have always been well masticated there is no physiological demand for a passage larger than that provided by the orifice of the diaphragm. An earlier and more acute incident would probably have occurred had the patient been unfortunate enough to swallow a foreign body which in normal people is usually accommodated easily by the pyloric canal. That the obstruction had been present for some appreciable time is suggested by the evident muscular hypertrophy of the stomach wall proximal to the diaphragm and the marked absence of hypertrophy between it and the pyloric sphincter. Radiographs (*Figs.* 323, 324), taken elsewhere two years earlier, show the diaphragm to be present at that time. The absence of any gastric delay on barium-meal examination is consistent with a partial obstruction which does not, however, prevent the passage of semi-fluid or fluid contents. The marginal inflammation of the orifice has probably precipitated the obstructive symptoms and was no doubt associated with the coexistent gastric ulcer.

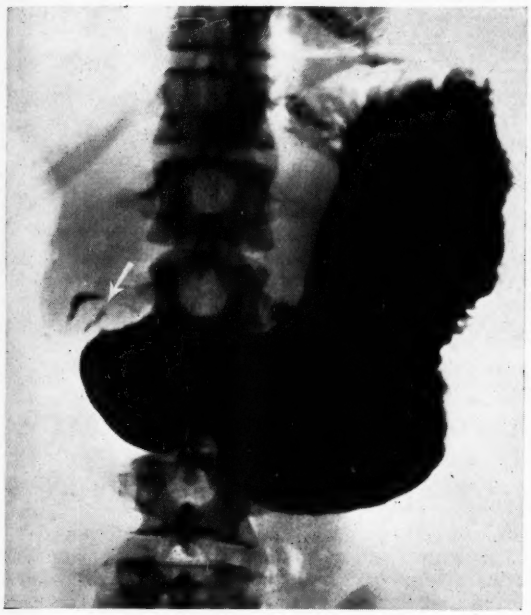

FIG. 323.—Barium-meal radiograph to show barium held up by diaphragm.

FIG. 324.—Barium-meal radiograph to show barium held up distal to the diaphragm.

The possibility of such an obstructing diaphragm being acquired must be considered. Redundancy of gastric mucosa may give rise to misleading radiological findings on barium-meal studies. Ricketts, Kirsner, and Palmer, 1947, describe three such cases of hypertrophied gastric rugæ which were mistaken for carcinoma of the pyloric antrum on barium-meal examination. The rugæ disappeared on insufflation with air and could not be seen on gastroscopy. They discuss the aetiology and favour the possibility of an underlying spasmodic factor of the muscular wall rather than an infective origin. Another interesting case has been described (Jenkinson and Latteier, 1943) where a filling defect of the pars pylorica was found on two successive radiographic studies, both of which examinations were preceded by atropine medication, and on both occasions the lesion was indistinguishable from a carcinoma. Gastroscopy findings were equivocal, the pylorus not being visible owing to a thickened fold of mucosa beyond the angularis on the lesser curvature. At operation the pyloric antrum

appeared normal to inspection, but palpation gave the impression of sustained localized gastrospasm. This spasm, however, underwent spontaneous relaxation after an interval of several minutes. The authors cite a similar case which was actually resected and where subsequent examination of the specimen revealed no abnormal pathology. They ascribe such conditions to " muscular mucosal spasm " and postulate some inco-ordination of the autonomic nervous supply. Forsell (1923) discusses at length the formation of rugæ and the mechanism and movement of mucous membranes. It might be argued that following such sustained muscular contraction of the antrum a permanent deformity of the mucosa may result and give rise to an obstructing diaphragm. These redundant mucosal rugæ of the pyloric antrum may be similar to those described by Eliason, Pendergrass, and Wright (1926), which cause prolapse of the mucosa into the pyloric canal and are sometimes associated with adult hypertrophy (Bockus, 1946) and old or recent duodenal ulceration.

Even if such a state of sustained muscular spasm in the region of the incisura does in fact take place, it would hardly seem justified to postulate that a permanent deformity can thereby result. The clearly defined and regular nature of the diaphragm, unassociated with any excess rugæ, would seem to be more in favour of its being congenital in origin.

SUMMARY

1. A case of partial atresia of the pyloric antrum by an obstructing mucosal diaphragm is described.

2. The possible factors in its aetiology are considered.

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TRAUMATIC FAT NECROSIS IN GREATER OMENTUM

WITH CLINICAL AND RADIOGRAPHICAL FEATURES SIMULATING CARCINOMA OF THE SIGMOID COLON

BY S. L. SPENCER, SYDNEY, N.S.W.

'TRAUMATIC fat necrosis' is most commonly reported in the female breast, where it may give rise to a mass which resembles, sometimes very closely, a scirrhous carcinoma in that organ.

It is the purpose of this communication to record an instance where traumatic fat necrosis involved the greater omentum and gave rise to a lesion the clinical and radiographic characteristics of which were such that a provisional diagnosis of carcinoma of the sigmoid colon was made, and a corresponding prognosis given to the relatives.

Although fat necrosis in the omentum from the action of pancreatic enzymes in acute pancreatitis is not uncommon, a search of the available literature has failed to reveal any report of traumatic fat necrosis in the greater omentum. Of further interest is the medico-legal aspect, since the condition appeared after an injury sustained during the course of the patient's employment.

CASE REPORT

HISTORY.—W. C. McD., a man of 54, was employed in a country coalmine in N.S.W., and, while pushing a truck, had fallen, striking the left lower abdomen on some projection. Although pain was experienced in this region, the patient continued at work for three days, but then the pain became more severe and some scalding on micturition was noticed. His doctor ordered proseptasine, and the urinary symptoms disappeared. A swelling, which did not disappear on catheterization, then developed above and to the left of the symphysis pubis. The following investigations were undertaken at the local hospital: X-ray examinations of the stomach and duodenum and of the renal tract showed no abnormality. An opaque enema was administered and it was reported that there was considerable delay in the sigmoid colon, after which the barium had flowed freely to the cæcum. No comment was made as to the presence or absence of deformity and the radiologist did not venture to suggest what was causing the delay.

The patient was referred to the author, and came to Sydney nine weeks after the original accident.

ON EXAMINATION.—A man in good general physical condition, who related the history detailed above. He stated that he had had typhoid fever at the age of 16, but no other illnesses. He had noticed no bowel upset, and was uncertain whether he had lost any weight.

Palpation of the abdomen showed a rounded mass, with a diameter of about 1½–2 in., apparently situated deep to the muscles of the abdominal wall and just above the left external inguinal ring. It was firm, slightly tender, and capable of limited movement. The liver could not be felt, and rectal examination was negative. A provisional diagnosis was made of carcinoma of the sigmoid colon, and operation was advised. The patient asked whether the lump was due to his accident and whether he was entitled to workers' compensation. He was told that this appeared improbable.

AT OPERATION.—A week later, on July 10, 1945, a laparotomy was performed. A hard mass was found in the lower part of the greater omentum. The mass was firmly adherent to the parietal peritoneum in the region where it had been felt on clinical examination, and the omentum was drawn tightly across the sigmoid colon, compressing it somewhat against the brim of the pelvis. The omental mass could not be separated from the parietal peritoneum, so the latter was removed with it, the bare area being covered by undermining and sliding flaps of the adjacent peritoneum.

The patient's post-operative progress was uneventful, and when written to in January, 1948, two and a half years later, he wrote in reply: "My condition since the operation has been most satisfactory in all respects, and I have suffered in no way, whatsoever, as a result."

THE SPECIMEN.—The tissue removed was submitted to Dr. Emily Day, and her report reads as follows: "The specimen consists of an irregular mass of fatty tissue. In the central part there is a dense golden yellow area. Part of this has been taken for examination.

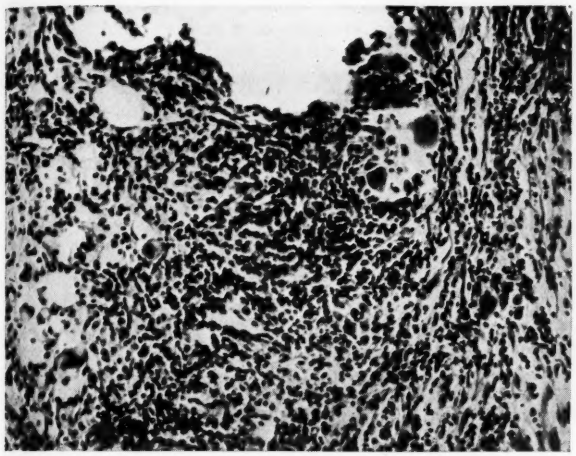

FIG. 325.—Granulation tissue containing lymphocytes and plasma cells occupies most of the field. Near the left border several round spaces represent remaining adipose tissue cells; adjacent large cells of irregular shape with vacuolated cytoplasm are lipoid-laden histiocytes. Multinucleated giant cells of foreign-body type are fairly numerous. (× 112.)

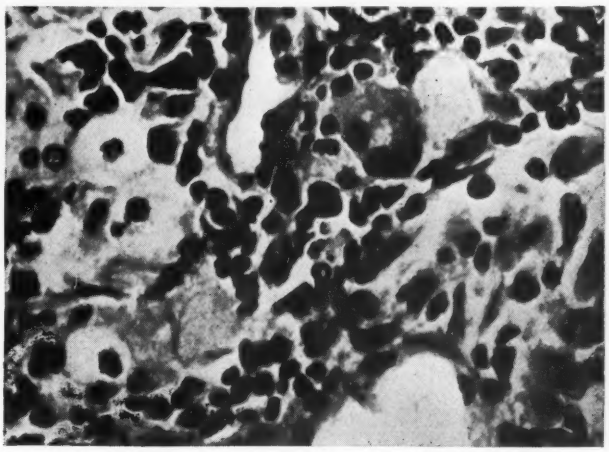

FIG. 326.—High-power photomicrograph showing details of giant cell and lipoid-laden histiocytes. (× 600.)

"*Microscopical*: The outstanding feature of the sections is the large number of histiocytes with foamy cytoplasm containing lipoid. Foreign-body giant cells, small round cells, and fibrocytes are also present. There is no evidence of new growth (*Figs.* 325, 326).

"*Diagnosis*: Omentum-fat necrosis."

The patient was advised of the final diagnosis, and a report forwarded to the Insurers, who accepted liability for compensation.

DISCUSSION

That fat in the animal or human body is unstable and may break down into glycerin and fatty acid is well known. It is probable that one or a combination of many factors may precipitate this change, but it has been observed to follow mechanical injury with such frequency that the term 'traumatic fat necrosis' has become widely used. The term is not a satisfactory one, for two reasons: first, what appear to be identical lesions may develop in a proportion of patients (possibly in the vicinity of 50 per cent), in whom no history of trauma can be elicited; secondly, the pathological condition is not a necrosis, but a saponification of the freed fatty acids, which provokes in the surrounding tissues a reaction comparable with that caused by foreign material.

'Traumatic fat necrosis' is essentially benign, and is usually without danger to the patient, and its most serious feature is that its clinical resemblance to carcinoma of the breast may lead to the performance of an unnecessarily radical and mutilating operation. It is not without interest that the same pathological process may be encountered in the greater omentum, and that here, too, it may provide a diagnostic pitfall.

I am indebted to Dr. A. A. Palmer for the photomicrographs and their descriptive captions.

ACUTE INTESTINAL OBSTRUCTION DUE TO LYMPHOSARCOMA OF THE ILEUM IN A CHILD AGED 5 YEARS

BY CONSTANCE M. OTTLEY, HOVE

CASE REPORT

A boy aged 5 years was sent into the New Sussex Hospital on the evening of June 5, 1945, with a diagnosis of acute appendicitis. His mother stated that he had complained since early that morning of colicky pains in the centre of the abdomen. He had vomited three times during the day and had not had his bowels opened. During the previous two years he had occasionally complained of similar pain, but the attacks had never been so severe as the present one.

The child was rather thin, but did not look ill. The temperature was 98°, the pulse-rate 110, the tongue moist and slightly furred. Examination of the abdomen disclosed a visible, firm, tender lump in the right iliac fossa. There was slight muscle guarding and the abdomen did not move on respiration. Physical examination was otherwise negative.

The findings seemed to me to be consistent with a well-shut-off 'acute appendix', and I decided to operate that evening.

AT OPERATION.—Through a lower right paramedian incision the mass was explored and found to consist of the last few inches of the ileum, which were dilated to almost adult diameter and contained what felt like a chain of large beads with a larger soft mass at its distal

end. In the belief that all the objects felt were foreign bodies the loop of bowel was packed off and an incision was made into it. The 'necklace' was then seen to consist of twelve raw green gooseberries lying one above the other, and the larger mass, of which the lower end lay about 2 in. above the ileocæcal valve, to be composed not, as had been expected, of hair or other swallowed material, but of soft, friable, vascular tissue. The growth had almost completely filled up the lumen of the bowel, and the gooseberries, unable to pass through the narrowed channel, had proved the last straw. The bowel wall was not thickened and the growth appeared to be confined within the muscular coat. No enlarged glands and no metastases in other organs could be discovered, and no fluid was present in the peritoneal cavity.

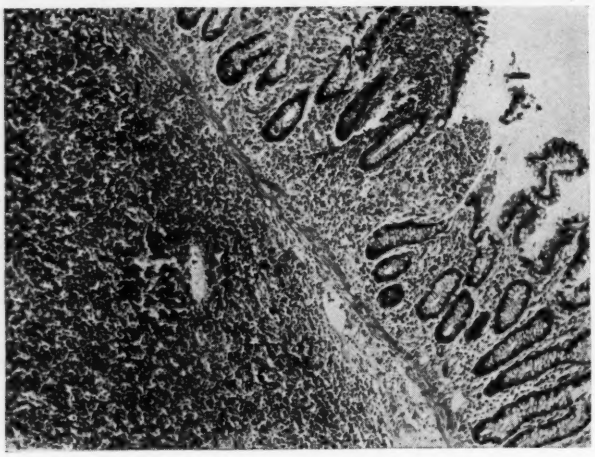

FIG. 327.—Low-power view showing replacement of muscle coat by tumour tissue and invasion of the mucosa. (× 45.)

The gooseberries having been removed, the small incision in the wall of the bowel was stitched up and the loop containing the tumour excised, with a good margin of healthy tissue. Just enough of the ileum was left above the ileocæcal valve for an end-to-end anastomosis to be made between the two cut ends of the loop.

PROGRESS.—The boy made a good recovery, though healing of the wound was delayed by his taking out the skin suture with his fingers when the nurses were not looking, and separating the skin edges. He was sent home with the wound healed on June 30.

Microscopical examination of the tumour shows it to be a lymphosarcoma widely infiltrating the muscular coat of the bowel (*Figs.* 327, 328).

The child was seen at three-monthly intervals and for eighteen months remained well and gained weight. In January, 1947, however, he began again to get attacks of abdominal pain and vomiting; he lost weight and was easily tired. Examination on Jan. 18 disclosed a slightly distended abdomen and a mass in the right iliac fossa. He had lost 1½ lb. in weight since his last attendance on Nov. 2. He was re-admitted to hospital, where exploratory laparotomy confirmed that recurrence had taken place; a mass of growth was present in the retrocæcal connective tissue. As soon as the wound was healed the child was transferred to the Royal Sussex County Hospital for a course of X-ray therapy, which was repeated six months later. The palpable mass almost disappeared, but the improvement was very transient; by the end of the year the mass was increasing in size and the child was obviously going downhill. He ceased to be able to attend as an out-patient, and died on Oct. 19, 1948.

AT AUTOPSY.—I am indebted to Dr. Blades for the following necropsy report:—

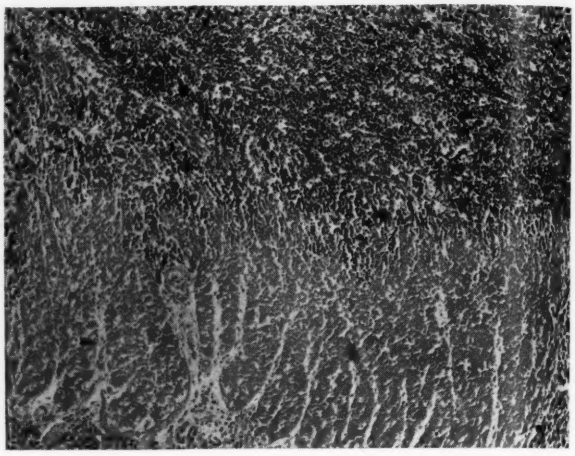

FIG. 328.—Low-power view showing extensive infiltration of the outer muscle coat. (× 45.)

External appearance: A very wasted boy with an ulcerated mass, 4 in. across, protruding through the skin of the lower abdomen on the right side.

Internal appearance: Alimentary system—the mouth, œsophagus, and stomach were normal. The duodenum passed behind the upper end of the mass, which occupied the whole of the right side of the abdomen below the liver and extended across to the left in the pelvis. The upper part of the small intestine was normal for a distance of about 6 in., after which mesentery and gut disappeared into solid tumour. The lumen of the intestine was continued through the tumour (which appeared to consist of gross thickening of the gut wall), and re-emerged about halfway up the ascending colon. At this point a diverticulum was present, admitting a finger and passing back to the posterior abdominal wall. The remainder of the large intestine was traceable, though bound to the mass in the pelvis. The tumour was white, opaque, and of a firm consistence; it had attached itself recently to the pelvis, posterior abdominal wall, and various viscera, but appeared to be essentially a thickening of the gut from the ileum to the ascending colon. There was no fluid in the peritoneum. There were no secondaries in the liver or peritoneum. No enlarged lymphatic glands were present. The lungs, cardiovascular system, kidneys, and suprarenals were normal. Spleen of septic type. Report of section: Lymphosarcoma or anaplastic sarcoma; probably the former.

REVIEWS AND NOTICES OF BOOKS

Advances in Surgery. Under the Direction of an Editorial Board (Chairman: WILLIAM DE WITT ANDRUS). 6 × 9 in. Volume I. Pp. 554 + xii, with numerous illustrations. 1949. New York and London: Interscience Publishers Ltd. 66s.

THIS volume, the first of a series, contains seven comprehensive articles dealing with different aspects of surgery, each written by an expert in his subject.

The first article deals with recent advances in traumatic shock; it is written by Jacob Fine, of Harvard, and gives the reader an up-to-date picture of the condition as we know it to-day. Warren Cole, of Chicago, writes on strictures of the common bile-duct, a subject he has made peculiarly his own.

Professor J. Z. Young, of University College, contributes by far the best article in the volume; his subject is factors influencing the regeneration of nerves. This article is profusely illustrated with excellent microphotographs and is a model of conciseness. The book is worth possessing for this article alone. J. E. Rhoads and H. A. Zintel discuss the use of antibodies in surgery, while Lingley writes on the immersion foot syndrome. A. H. Blakemore and J. W. Lord contribute an interesting chapter on blood-vessel anastomosis by means of a non-suture vitallium tube method; and B. L. Coley and N. L. Higinbotham deal with tumours of bone.

A very useful set of articles complete with up-to-date references.

Functional Localization in the Frontal Lobes and Cerebellum. With Particular Reference to the Operation of Frontal Lobotomy. By JOHN F. FULTON, O.B.E., M.D., D.Sc., LL.D. (Birm.). Being the William Withering Memorial Lectures delivered at the Birmingham Medical School, 1948. 5⅜ × 8½ in. Pp. 140 + xii, with 53 illustrations. 1949. London: Oxford University Press. 15s.

THIS interesting monograph contains the William Withering Memorial Lectures delivered by Professor Fulton at the Birmingham Medical School in 1948. The functions of the prefrontal cortex in the light of recent discoveries in the clinical, anatomical, and experimental fields are well described, especially the control of emotion and social behaviour. In the treatment of some of the psychoses the modern operation of lobotomy is discussed, and the author considers that this operation may be replaced at a future date by a less severe procedure.

A most interesting and fascinating book which will stimulate the curiosity and imagination of all medical men and women who have to treat neurological and psychotic patients.

Tumours of Bone. By CHARLES F. GESCHICKTER, M.D., Professor of Pathology, Georgetown University Medical School, etc., and MURRAY M. COPELAND, M.D., Professor of Oncology, Georgetown University Medical School, etc. Third edition. 6⅞ × 10 in. Pp. 810 + xviii, with 642 illustrations. 1949. Philadelphia and London: J. B. Lippincott Co. 105s.

THE new third edition of this important work has been very thoroughly revised and much new matter has been added. It is some twelve years since the second edition was published, and in that interval quite a considerable number of fresh entities have come to light in the field of bone pathology.

The new edition contains some new chapters on fibrous dysplasia, osteoid osteoma, tumours of the spine, and rare diseases of bone.

The vast experience of the two authors in the field of bone pathology has resulted in a mellowed description of every aspect of tumours arising in the skeletal system.

It is refreshing to find a book in which the clinical side is incorporated in the pathological, and clinical photographs are correlated with X-ray pictures, and these in turn with pictures of the gross and microscopical pathology. This is the way pathology should be taught in every medical school, but, alas, how often such a scheme is wanting!

The outstanding feature of this book is the excellence of the illustrations, both X-ray and pathological. We have only one criticism, and that is the omission of the actual magnification in the microphotographs. Every microphotograph should have the magnification clearly inserted, for it gives the reader a much better idea of the microscopical field.

This new edition should appeal to students and graduates alike, and it will take its place as the reference volume on the subject of bone tumours for several years to come.

Manual of Medical Emergencies. By S. C. CULLEN, M.D., Professor of Surgery; Chairman, Division of Anæsthesiology, State University of Iowa College of Medicine, and E. G. Gross, M.D., Professor and Head of Department of Pharmacology, State University of Iowa College of Medicine. 4¾ × 7 in. Pp. 267, with 29 illustrations. 1949. London: H. K. Lewis & Co. Ltd. 21s.

THE average medical man is apt to consider that medical emergencies are few and far between, but the authors of this small manual have proved that this is quite incorrect. The book is devoted to the treatment of medical emergencies.

The circulatory emergencies form a large group, and an all too common group, of cases. Angina pectoris, coronary occlusion, cardiac dysrhythmias, and the cerebrovascular accident are met with daily. The value of nitrites, morphine, and the barbiturates is set out in a clear and lucid manner.

Acute poisoning is dealt with in a very practical way.

Head injuries, allergic disturbances, and the various comas are all treated in a very sensible manner.

One would have thought that the treatment of burns was a surgical problem and not a medical one.

The book fulfils its purpose and is a useful manual for the student and the modern nurse.

A Companion in Surgical Studies. By IAN AIRD, Ch.M., F.R.C.S., Professor of Surgery in the University of London; Director of the Surgical Unit, Postgraduate Medical School of London. 7¼ × 9⅝ in. Pp. 1060 + viii. 1949. Edinburgh: E. & S. Livingstone Ltd. 63s.

"EVERY age hath its book", and this may well be said of Professor Aird's *Companion in Surgical Studies*. This large volume is unique in many respects, for it is written by a single author, there are no illustrations, and it covers the whole field of surgery with the exception of orthopædics and plastic surgery. The presentation is pleasing and the style decisive and lucid.

The author appears to know just what is required and has the happy knack of knowing what to exclude from the text. His vast knowledge of surgery, his clinical acumen, and his practical capabilities are all clearly evident in this book. The volume is encyclopædic and the author has covered the whole range of common and rare surgical diseases and conditions.

One misses illustrations in this book, and although the author considers that " visual memory enchains the intellect ", yet one illustration well placed may save pages of descriptive text.

The senior student and the post-graduate will find in this volume a mine of information, while the surgeon will not turn in vain to its pages when confronted with a difficult surgical problem.

We hope that the second edition will be published in two volumes so that the reader will be able to handle it more easily.

A book that is a credit to the surgical literature of England.

Illustrations of Surgical Treatment. Instruments and Appliances. By ERIC L. FARQUHARSON, M.D., F.R.C.S. (Eng. and Edin.), Assistant Surgeon, Royal Infirmary, Edinburgh. With a Foreword by the late Sir JOHN FRASER, Bt. Third edition. 6 × 9¾ in. Pp. 391 + xii, with 319 illustrations and 61 plates. 1949. Edinburgh: E. & S. Livingstone Ltd. 25s.

THE third edition of this practical guide to the use of surgical instruments and appliances has been very thoroughly revised and brought up to date.

It should prove of real value to students and nurses who want to gain a knowledge of the practical side of surgery.

The illustrations are excellent.

Atlas of Plastic Surgery. By MORTON I. BERSON, M.D., formerly Director of the Department of Plastic and Reconstructive Surgery, The Downtown Hospital and Pan-American Clinic, New York City. 7⅞ × 10¾ in. Pp. 304 + xii, fully illustrated. 1949. London: Wm. Heinemann (Medical Books) Ltd. 75s.

SOME clue as to the difference between an atlas and a text-book may perhaps be obtained from the arrangement of the text and illustrations of this book in parallel columns facing one another. For such, a typically American, but none the less valid, claim is made that the reader thereby avoids " the necessity of time-wasting re-orientation at each shift of attention between text and pictures ". The result is pleasing, as text and figures are easily followed and the clarity of each is praiseworthy. The anatomical diagrams are particularly commendable and the technique of plastic surgery is well illustrated throughout. Some idea of the scope of the work may be gathered from the fact that sections are included on the repair of skull defects, mammaplasty, cleft lip and palate, face lifting, cauliflower ear, rhinophyma, and the correction of facial palsy. Proof reading on the whole has been well done, but ' gage ' on page 59 should obviously be ' gouge '. Some patients we are told (p. 91) bring photographs of ' movie stars ' with them and a request to have their noses fashioned to resemble those on the photographs, but the author remarks that by constructing a mechanical nose on the patient's mask it can be demonstrated that the desired nose is disproportionate to the rest of the features. We might well comment, " *vanitas vanitatum, omnia vanitas* "!

The book is a valuable guide in the fields of reconstructive and reparative surgery, and as such should prove attractive to a great many surgeons.

Text-book of Surgery. By PATRICK KIELY, B.Sc., M.D., M.Ch. (N.U.I.), F.R.C.S. (Eng.), Professor of Surgery, University College, Cork; etc. 6 × 9¾ in. Pp. 1184 + vii, with 611 illustrations. 1949. London: H. K. Lewis & Co. Ltd. 45s.

THIS single volume of nearly 1200 pages emanates from the surgical school of Cork and is a monumental work for a single author. Professor Kiely has in fact enlisted the aid of a colleague (Professor O'Hea Cussen) to write the chapters on the " Ear, Nose, and Throat " and the " Eye ", and frankly admits that he has drawn largely on other text-books of surgery for much of his information. Particularly is this so in the chapters on Fractures and Orthopædics, where due acknowledgement is given to Watson-Jones and Mercer respectively.

This book is a composite development of the author's lectures to students, and in this light is on the whole more than adequate. The paragraph headings and the classification tables are such as are dear to the student's heart. Some are really excellent, as, for instance, the descriptions of neck swellings and of surgical diseases of the spleen. Throughout stress is laid on the clinical pictures and both pathology and treatment suffer correspondingly. Operative technique is deliberately omitted —which is no fault, but many modern methods now fully accepted by the profession are mentioned as though still in the trial stage. The impression is almost given that even penicillin comes into this category.

Although in most places satisfactorily succinct, the book is in no sense a synopsis. It contains brief allusions to many rare conditions, and one finds mention of the role of sympathetic surgery, of developments in cardiac surgery, of disk displacement, and of diseases of the thymus. The surgical aspects of hypertension, the anticoagulants, and the use of vagotomy appear to have been overlooked.

But there is an old-world atmosphere about the book which keeps cropping up. Examples are the treatment of erysipelas by prontosil, the remark that in the treatment of syphilis " experience with penicillin . . . . is most encouraging, but it is too soon yet to make a final pronouncement ", the treatment of burns with picric acid, the statement that parotid tumours (classed amongst the teratomata) contain cartilage, and the author's firm belief in the necessity for primary operative treatment in acute osteomyelitis. Again, he advocates gastro-enterostomy for duodenal ulcer, though admittedly allowing that many surgeons now employ partial gastrectomy. Surely the intestinal flogging methods for the treatment of paralytic ileus are things of the past.

Both anæsthesia and gynæcology are dealt with by the author—the former rather too briefly to be of great value and the latter rather too extensively for a text-book of general surgery. The book is copiously illustrated, many of the drawings being semidiagrammatic. Some of the radiographic reproductions (e.g., Fig. 271) are of little value, but in general the publisher's work is highly creditable. The index is not without flaws.

This may seem rather carping criticism of a very comprehensive students' text-book. There is much in it that is excellent. It is readable, commendably well set-out, and quite original in its handling of various matters, but it nevertheless lacks that touch which might make it a serious competitor of its various English counterparts.

Clinical Aspects and Treatment of Surgical Infections. By FRANK LAMONT MELENEY, M.D., F.A.C.S., Associate Professor of Clinical Surgery, College of Physicians and Surgeons, Columbia University; etc. With a Foreword by ALLEN O. WHIPPLE, M.D. 6⅜ × 9⅝ in. Pp. 840 + xiii, with 287 illustrations. 1949. Philadelphia and London: W. B. Saunders Co. 60s.

THIS is an astonishing book. A production from the pen of one with the world-wide reputation of Dr. Frank Meleney and emanating from the Presbyterian Hospital of New York might well be expected to be something beyond the ordinary. Such a remark alone would express very faint appreciation of a really remarkable work. Similarly, simply to state that the book consists

of some 800 pages, interspersed with no less than 300 excellent illustrations, describes hundreds of cases (both those of the author and his colleagues), includes at the end of each chapter an extensive bibliography (not limited only to American authors), and is completed by a most exhaustive and accurate index—would be but to catalogue the dry bones on which the author has hung a mass of interesting, practical, and stimulating facts.

So wide a field is covered—" all areas, organs, and tissues infected with necrotizing and pyogenic organisms " —that each individual chapter is a relatively succinct and concise description of the problem under consideration.

Throughout, the work bears witness to the great value (stressed also in the preface by Dr. Whipple) of an intimate integration of a bacteriological laboratory with a surgical service. Certain particular problems stand out as affording major interest to the author. Two of these are obviously the " undermining burrowing ulcers " and " progressive bacterial synergistic gangrene ". It will be admitted that these conditions are rarely seen in practice to-day, but occasional cases do still crop up and, if nothing else, the descriptions of these form an historical annotation of the greatest value—a perfect pen-picture of the pre-antibiotic days.

Referring to antibiotics, the author has in his laboratory produced one which is peculiar to himself—Bacitracin. This substance is an antibiotic derived from bacteria, as against those from the various moulds. Bacitracin in the hands of its producer obviously produces dramatic and excellent results, but it is only fair to state (and let it be admitted there is no parsimony in the outlook of the author) that it has never reached the status of penicillin and streptomycin. Both these latter receive full credit in the book.

From the foregoing it will be obvious that the scope of this really epic work is entirely comprehensive, and it is therefore difficult to single out any particular chapter for especial mention, but undoubtedly one of the most informative is that on ulcerative colitis. Throughout, the wide experience of the author balances nicely the relative values of surgical and non-operative treatment, and in both fields of therapy brings out many individual practical points.

This is not a book to be read from cover to cover, though the reader will find temptation so to do, but it will provide full information for reference on any one aspect of surgical infections that may be required. It is well bound and excellently produced and offers more than good value for money.

Neurology. By ROY R. GRINKER, M.D., and PAUL C. BUCY, M.D., Chicago. Fourth edition. $6\frac{1}{2} \times 9\frac{7}{8}$ in. Pp. 1138 + ix, with 393 illustrations. 1949. Springfield, Ill.: Charles C. Thomas. (Oxford: Blackwell Scientific Publications.) 63s. net.

THE fourth edition of this large and comprehensive volume on neurology has been very thoroughly revised, and the addition of a joint author in Professor Bucy has certainly increased the value of the book, for he brings with him a vast knowledge of clinical neurology and neurosurgery.

The usefulness and popularity of this work can be judged by the large number of reprintings that have been required since the third edition was published in 1943.

The usefulness of this book is due to the direct and lucid form of diction, the excellence of the illustrations, and the up-to-date bibliography.

The authors write from ripe practical experience gained over many years of careful study of a vast amount of neurological material. They have the rare but happy knack of placing first things first, and bringing to light the important facts in neurology.

As a reference volume this book could not be bettered, for it covers the whole vast field of neurology in a very comprehensive way.

Perhaps the best section of the book is that devoted to intracranial tumours, for the difficult histopathology is made easy by the beautiful microphotographs of the different tumours of the brain and spinal cord.

Students and practitioners will not turn in vain to its pages when they want enlightenment on any neurological subject.

A very useful book and one that every neurosurgeon should have on his bookshelf.

An Account of the Schools of Surgery, Royal College of Surgeons, Dublin, 1789–1948. By J. D. H. WIDDESS, M.A. (Dublin), L.R.C.P. and S.I. With a Foreword by WILLIAM DOOLIN. $6\frac{5}{8} \times 9\frac{3}{4}$ in. Pp. 107 + viii, with 16 plates. 1949. Edinburgh: E. & S. Livingstone Ltd. 17s. 6d.

" THE history of medicine is in a sense the history of civilization. The state of medicine amongst a people is an index of its culture as much as its art and literature." —*David Riesman.*

In the Preface the author states that the book is an expansion of three lectures given to the students of the Royal College of Surgeons in Ireland so that they might learn something of its tradition—an example which might be emulated in other schools. This tradition is not only of local interest but must appeal to all who have to educate medical students in clinical medicine and surgery.

The author has been fortunate in having the encouragement of such a well-known medical historian as William Doolin to write the Foreword, which is an example of *multum in parvo* by its brevity and penetrating appreciation of the author and his book.

The Irish, out of their own resources, untouched by Rome, developed a remarkable indigenous culture. They elaborated a type of Christianity which they sent to Scotland, Northumbria, and Europe. The Italian Renaissance hardly crossed the Irish Sea, yet this culture was maintained.

During the medieval period and subsequently Universities with medical schools were founded in various towns in Italy, France, England, and Scotland which attracted students from all over the world.

Yet just when the influence of the Edinburgh School began to wane at the beginning of the nineteenth century that of Dublin sprang into prominence, due to a small group of men and especially to two, master and pupil—namely, Robert Graves (1796-1853) and William Stokes (1804–78). It is instructive to consider what modern medicine owes to the Dublin physicians and surgeons of this period. First and foremost they were all anatomists, normal and morbid, and it may be that the thorough grounding they received in this basic science played no small part in their later clinical acumen and successful bedside teaching.

Their knowledge of anatomy was helped by the ease with which the necessary material was obtained. As the author states: " Anatomy was the fundamental subject at this period. It was regarded by the College authorities as the fountain of medical knowledge ", and this is still the case no matter what the modern educationalist of medical students may think and argue. Exciting details are given as to the methods whereby the acquisition and disposal of this anatomical material was developed until the Anatomy Act of 1832 made legal provision for it.

Medicine owes to these clinicians of Dublin many things which are daily taught to all medical students throughout the world :—

Francis Rynd (1801–61) introduced hypodermic medication.

Abraham Colles (1773–1843) established Colles law in hereditary syphilis (1837); he described " the fracture of

the carpal extremity of the radius " (1814) and published other well-known papers and books, one of which, *A Treatise on Surgical Anatomy*, published in 1811, was probably " the first anatomical work designed on topographical lines ". In it he described Colles fascia and Colles ligament as original observations.

The author is, however, very wide of the mark when he states : " The first account of the spread of mammary cancer to adjoining lymphatic glands is given ". This spread to the lymphatic glands had been known since the time of Galen, and was accurately described by Ambroise Paré.

Colles was Professor of Anatomy and Surgery of the R.C.S. in Ireland from 1804–36. When he commenced teaching in 1800 there were 60 medical students in Dublin ; when he resigned the chair in 1836 there were 1000. By his foresight and persistence he convinced the authorities of the R.C.S. that chairs of chemistry, medical jurisprudence, comparative anatomy, and botany should be established in addition to those of surgery and medicine.

In 1841 the College made an historic appointment when they elected a professor of hygiene or political medicine. This was the first chair devoted to preventive medicine in Great Britain and Ireland.

William Wallace (1791–1837) introduced potassium iodide for the treatment of late manifestations of syphilis.

Robert Graves (1796–1853) published a description of hyperthyroidism in 1835 and it has never been surpassed. In 1848 he published his *Clinical Lectures* and thereby stimulated bedside teaching. He revolutionized the treatment of ' fever ' so much so that he wished his epitaph to be ' He fed fevers '.

William Stokes (1804–78) appreciated the importance of Laennec's discovery and published a treatise on the use of the stethoscope in 1825, before he had obtained any medical qualification.

This book raised a storm of controversy, but resulted in the wide adoption of the instrument by clinicians.

Laennec had interested himself in the description of the various physical signs detected ; Stokes correlated these with the symptoms. Together with Graves, Colles, and Corrigan, he founded the Pathological Society of Dublin in 1838, and with *Robert Adams* (1791–1873) he reported the first case of Stokes-Adams syndrome in 1846.

John Cheyne (1777–1836) described Cheyne-Stokes respiration in 1818 in the *Dublin Hospital Reports*.

Sir John Dominic Corrigan (1802–80) published in 1832 his paper " On permanent patency of the mouth of the aorta or inadequacy of the aortic valves ". He stated that when he applied for membership of the Royal College of Surgeons of England he was asked : " Are you the author of the essay on the patency of the aortic valves ? " His affirmative gained him immediate admission.

The above is no mean contribution to clinical medicine from one school in such a few years. The students went all over the world and spread this teaching and the methods whereby it was given. The majority of medical men in the armed services of the Crown in the Napoleonic wars received their training in Dublin, either in the schools of surgery founded by the R.C.S. or in the Dublin University. There were a number of private anatomy schools like those in England and Scotland which were all ultimately absorbed into that of the R.C.S., which was founded and ready for occupation in 1789.

This book can be safely recommended to every medical man as a stimulating essay on the development of his science and art, and the method of teaching it to students. He will learn that Dublin has more to offer to the medical world besides the teaching of midwifery in its famous Rotunda Hospital. The surgical school founded by the Royal College of Surgeons in Ireland illustrates by its work that " surgery engrossed in scientific investigation reserves insignia of greatness for those who find their satisfaction in service and in the search for the elusive secrets which benefit mankind ".

Fractures and Dislocations in General Practice. By JOHN HOSFORD, M.S. (Lond.), F.R.C.S., Surgeon to St. Bartholomew's Hospital. Second edition, revised by W. D. COLTART, M.B., B.S. (Lond.), F.R.C.S., Assistant Orthopædic Surgeon to St. Bartholomew's Hospital. $5\frac{1}{2} \times 8\frac{1}{2}$ in. Pp. 290 + x, with 87 illustrations. 1949. London : H. K. Lewis & Co. Ltd. 21s.

IT is just ten years since the first edition of this book appeared, and in that decade considerable advance has been made in the treatment of fractures. We are living in a traumatic age and the diagnosis and treatment of fractures is most important.

The new edition of this book has been carefully revised and new illustrations have been added. It should prove of real value to the student and practitioner, for it sets out in a clear way the treatment of fractures which may be met with in general practice.

Acute Injuries of the Head. Their Diagnosis, Treatment, Complications, and Sequels. By G. F. ROWBOTHAM, B.Sc. (Manch.), F.R.C.S. (Eng.), Surgeon in Charge, Department of Neurological Surgery, Newcastle General Hospital ; Lecturer in Neurosurgery, University of Durham ; etc. With a Foreword by Professor NORMAN M. DOTT. Third edition. $6\frac{3}{4} \times 9\frac{3}{4}$ in. Pp. 480 + xx, with 259 illustrations, 34 in colour. 1949. Edinburgh : E. & S. Livingstone Ltd. 35s.

IN developing into its third edition, this book has enlarged considerably, and its volume is now more than twice that in which it first appeared in 1942. It remains, however, entirely the work of Mr. G. F. Rowbotham and has not become a composite work with included chapters or articles by others as is so often the case in succeeding editions of an established book. Along with an increase in the amount of text there are more illustrations. Both maintain the high standard set by the first edition and serve to incorporate later war experience. This has particularly emphasized the value of early and complete surgery in cases of open head wounds and the importance of correct rehabilitation, and due regard is paid to both aspects of the subject. Mr. Rowbotham still supports the use of ammioplastin (p. 292) even though many surgeons, unable to substantiate the claims of its introducers, have abandoned this in preference for sheet fibrin products. A common error appears of page 40, where Queen Square appears as Queen's Square. There is little to criticize in this monograph and a great deal to commend. It is an admirable and clearly-written account of the subject, of which both its author and the publishing house which produces it may well be proud.

Clinical Methods in Surgery. By K. DAS, M.B. (Cal.), F.R.C.S. (Eng. and Edin.), Surgeon, R. G. Kar (Carmichael) Medical Hospitals and Medical College Hospitals, Government of Bengal. Second edition. $7 \times 9\frac{3}{4}$ in. Pp. 255 + viii, with 249 illustrations. 1949. Calcutta : City Book Co. (London : H. K. Lewis & Co. Ltd.). 35s.

THAT a second edition of a book is required within a year is clear proof of its usefulness. Mr. Das is to be congratulated on his efforts to bring clinical teaching to the forefront in the teaching of surgery to-day. The vast sub-continent of India has enormous clinical material, and Mr. Das has commenced to tap this source and we hope he will continue in his efforts.

The Story of the Johns Hopkins. Four Great Doctors and the Medical School They Created. By BERTRAM M. BERNHEIM, M.D. $4\frac{7}{8} \times 7\frac{1}{4}$ in. Pp. 274 + xiv. Illustrated. 1949. Kingswood, Surrey: The World's Work (1913) Ltd. 12s. 6d.

ANYONE who has read *A Surgeon's Domain* will always wish for more from the same author. Professor Bernheim has given us of his best in this second book of his, the inner history of the great American Medical School, Johns Hopkins.

In England and America the name Johns Hopkins is the symbol of the highest standards in medical education and teaching.

It is curious to note that Johns Hopkins was created in the atmosphere of four great men, namely, Welch, Osler, Halsted, and Kelley. These men were known the world over and Osler came over to Oxford later and helped to create a medical school in that ancient University city.

Professor Bernheim is fortunate in his background, for he went to Hopkins soon after the great medical centre got under way. As an undergraduate, house surgeon, and now Professor of Surgery, he is very well versed in all that has happened at this great institution.

This book tells of the great efforts of the men who not only made the name of Johns Hopkins great but also kept the standard of medical education high and were ever to the front in developing new surgical techniques and passing them on to the younger group of medical men.

An excellent book which should appeal to all surgeons in the British Isles.

The Temporal Bone and the Ear. By THEODORE H. BAST, A.B., Ph.D., Professor of Anatomy, University of Wisconsin, and BARRY J. ANSON, M.A., Ph.D., Professor of Anatomy, Northwestern University Medical School. Pp. 478 + xviii, with 165 illustrations. 1949. Springfield, Ill.: Charles C. Thomas. (Oxford: Blackwell Scientific Publications.) 60s.

EVER since 1824, when Flourens carried out experiments on the semicircular canals of pigeons which led to the recognition of audition and equilibration as distinct functions of the inner ear, detailed studies of the minute anatomy have been made in an attempt to cast further light on the functions of its somewhat complex arrangement. Among the latest are those of the authors of this book, who give a detailed and well-illustrated account of the middle and inner ear, including its development. They begin with the labyrinth, which is the only part of the internal ear present in the early stages of the fœtus and have concentrated their investigations on the otic labyrinth (containing endolymph or otic fluid) and the periotic labyrinth (containing the perilymph or periotic fluid), and the otic capsule, as they prefer to designate the membranous and bony parts of the labyrinth respectively. Among many excellent illustrations are some familiar ones from Gray's and Cunningham's textbooks.

Some idea of the scope of the book may be gathered from some of the chapter headings, such as, The Temporal Bone and the Ear, The Otic Labyrinth, The Periotic Labyrinth, The Otic Capsule, The Origin and Development of the Middle Ear and Related Air Spaces, and The Auditory Ossicles. In many respects the most fascinating chapter, however, is that entitled, A Historical Survey of the Structure and Function of the Internal Ear, in which theories of hearing are discussed from the time of the early Greeks up to to-day. Nevertheless, at the end of the chapter the authors repeat the question with which they open it, namely, "How do we hear?" and have to conclude that it is still unanswered.

Bibliographies are placed after each chapter, and these are for the most part representative, but names we sought in vain were Charles Ballance and J. Ernest Frazer. Printing is clear on highly glazed paper and the illustrations are excellent. Fig. 5 on page 11 is especially deserving of notice in showing the relationships between perilymph, endolymph, and the cerebrospinal fluid. Useful summaries conclude certain of the chapters. The book is dedicated to John Gordon Wilson, M.D., Emeritus Professor of Otolaryngology in Northwestern University Medical School, and is a worthy tribute to him by his younger colleagues, who have performed a valuable service in their production of this comprehensive monograph.

A Practice of Orthopædic Surgery. By T. P. MCMURRAY, C.B.E., M.B., M.Ch., F.R.C.S. (Edin.), Professor of Orthopædic Surgery, Liverpool University; etc. Third edition. $5\frac{3}{8} \times 8\frac{1}{2}$ in. Pp. 444 + viii, with 191 illustrations. 1949. London: Edward Arnold & Co. 30s.

AFTER six years, during which period there have been three reprints of this book, a third edition has appeared.

There are no drastic changes: indeed they are minimal. A section on coracoid osteotomy has been added, the only effect of which is to add to the confusion in the treatment of recurrent dislocation of the shoulder —when most of us thought that increasing recognition of the supremacy of Bankart's work was dispelling the artificial fog that had for too long surrounded an essentially simple subject. There is a valuable section on intervertebral disk lesions, which are also recognized as a (but not *the*) curse of sciatic scoliosis. Cautious approval is expressed of ganglionectomy in the treatment of severe causalgia, and of autogenous grafting for the repair of large gaps in peripheral nerves. The operative treatment of brachial plexus injuries still finds a place in the book; in view of the depressing pathology of traction injuries and the almost uniform futility of operations carried out by very competent men during the war, it would be interesting to know the evidence on which the author's recommendations are based. It is good to find that Professor McMurray insists on the administration of large doses of penicillin, over a period of three weeks, in the treatment of acute osteomyelitis; and that he still advocates metaphysial drilling, though surely he goes a little too far in always advising it in patients more than one year of age.

Two irritating misprints persist; epiphyseodesis is still called epiphyseolysis; and Perthes, although partially degallicized (he was Perthé in the second edition), again appears incorrectly as Perthe, except on page 344 where the acute accent is retained.

Cunningham's Manual of Practical Anatomy. Revised and edited by JAMES COUPER BRASH, M.C., M.A., M.D., F.R.C.S. (Edin.), F.R.S.E., Professor of Anatomy, University of Edinburgh. Eleventh edition. $4\frac{7}{8} \times 7\frac{1}{4}$ in. Vol. II: Thorax and Abdomen. Pp. 488 + x, with 238 illustrations. Vol. III: Head and Neck; Brain. Pp. 513 + x, with 230 illustrations (including 16 plates), many coloured. 1949. London: Oxford University Press. Each volume, 21s.

VOLUME II: The second volume of this well-known manual is devoted to the practical anatomy of the thorax and abdomen. It has been very thoroughly revised and many new illustrations have been added. We are glad to see a definite tendency to place applied or surgical anatomy in its proper place.

The volume is of handy size and convenient for the pocket. It still remains in our opinion the best book for the student and practitioner on practical anatomy.

VOLUME III: To many the anatomy of the head and neck, and especially the brain, is by far the most difficult region of the body to remember. Yet, when the practical

side is described as it is in this volume, the whole subject appears simple and of real value not only to the student but also to the surgeon who wishes to refresh his memory. Professor Brash has incorporated some new illustrations and has cut out some of the descriptive details which, in his opinion, were not required.

The new edition is a worthy successor to the numerous ones which preceded it, and the book will not have a rival as far as medical students in this country are concerned.

Lawson Tait, 1845–1899. By I. HARVEY FLACK, M.D. $4\frac{3}{4} \times 7\frac{1}{4}$ in. Pp. 148 + viii, with 12 plates. 1949. London: William Heinemann (Medical Books) Ltd. 17s. 6d.

THIS has been a somewhat difficult biography to assess. The subject—Tait's contribution to the progress of British surgery—was originally selected by the author as the leitmotiv for the Thomas Vicary Lecture delivered at the Royal College of Surgeons in 1947. The notes for the Lecture were subsequently expanded into the book under review. The reviewer's difficulty lies in the query: was such expansion really necessary?

The principal accomplishment of this record will have been to lay for ever the rumours as to Tait's birth—rumours which, self-reliant and coarse-grained as he was, he himself was never at pains to deny. Stewart McKay—whose larger biography appeared in 1922—was firmly of the belief that Tait had been the natural son of Sir James Young Simpson, and McKay's categorical assertion has been mainly responsible for that belief being widely held in the past quarter of a century. After a thorough sifting of all available sources of information, Mr. Flack now shows conclusively that this widely accredited rumour has been in fact only a rumour, with not a shred of credible evidence to support it. Of Tait's energy, self-confidence, pugnacity, and tenacity of purpose, as well as of his consummate craftsmanship as an operator, his biographer here conveys a wholly convincing impression. These qualities rapidly placed Tait, *primus inter pares*, to the fore among the pioneers of abdominal surgery at a time when the work done in the London schools lagged far behind that of the provincial centres. But, "Birmingham Radical" in more senses than one, his wrong-headed methods in debate, even when he was in the right of the argument, often contributed more heat than light to the subject under discussion, tending, such is our poor human nature, to retard rather than to facilitate progress in the field he had made so peculiarly his own. In the end these very qualities were to lead directly to his pathetically tragic fall.

But even when his fortunes stood at their lowest ebb he could look back on two outstanding personal contributions to the advancement of British surgery, the lack of which, but for his acumen and thrust, would have delayed that progress for a considerable time: during his meteoric career he had blazed the trail for the the great line of abdominal surgeons who followed, with Rutherford Morison and Moynihan, provincials like himself, at their head, and his individual effort had saved the Medical Defence Union from premature fatality even at its birth.

"It is the personal alone, sir, which interests mankind." So long as Dr. Johnson's aphorism holds true, so long opinions will vary as to Tait's merit and character. Mr. Flack's portrait is that of neither the incorrigible sinner nor yet that of the impeccable saint: he presents him as a "completely ambivalent character" with all the faults of his virtues; not a 'genius', but with sufficient of the qualities of genius to have blazed the trail he did in the face of prejudice and opposition often begotten of his own ruthlessness. Modestly, Mr. Flack expresses regret that this has been "very much a one-man story". His hero, it is true, is of almost Gargantuan proportions, but he has been set in an admirable perspective of the ungentle professional days in which he lived.

Minor Surgery. Edited by Sir HENEAGE OGILVIE, K.B.E., D.M., M.Ch., F.R.C.S., and WILLIAM A. R. THOMSON, M.D. With an Introduction by LORD WEBB-JOHNSON, K.C.V.O., C.B.E., D.S.O., T.D. Second edition. $5\frac{3}{4} \times 8\frac{1}{2}$ in. Pp. 192 + xxiv, with 34 illustrations. 1949. For *The Practitioner* by Eyre & Spottiswoode.

DEFINITION is often the downfall of author, editor, and even reviewer. Only a comfortably elastic outlook would permit *Minor Surgery* being allowed as a correct title for this undoubtedly excellent little book. Many of the authors of the articles in it have obviously appreciated this fact.

Let it be said, it in no way detracts from its value but, perhaps, should increase its scope. Originally, when first published six years ago it was a compendium of monthly articles written for the *Practitioner*; it has now been thoroughly revised and added to. With eminent authors from London, Edinburgh, Oxford, Manchester, and Durham, it represents a very good cross-cut of surgical opinion in the country, and the amount of good sound practical knowledge contained in its less than 200 pages is astonishing. But apart from treatment, very adequate consideration is given to the pathology of conditions considered, and aetiology is not omitted. It is stimulating to note the stress that is laid throughout on prophylaxis, and the absence of any but minor overlaps and differences of opinion points to careful editing.

Lord Webb-Johnson's Introduction sets the tone of the whole book—succinct, clear, and practical. It is somewhat invidious to pick out individual chapters when all are good, but in that they adhere most closely to the title and include a large amount of valuable information in the minimum of space, perhaps the articles of Gabriel (Rectum), Clifford Morson (Genito-urinary Surgery), Girdlestone (Strains and Sprains), and Lake (The Hand) stand out. Coyte's short article on Trusses and Belts fills a very definite want and the new chapter on Chemotherapy (Thomson) is an excellent prècis of the subject. It is not without interest to consider that the antibiotics were virtually unknown when the first edition was produced, and so rapid still are advances in this field that one can already query the author's remark that streptomycin is "only in the stage of clinical trial".

The references at the end of many of the chapters are valuable, and the index is particularly good. The same cannot be said for the illustrations. Such as there are, no fault can be found with them, but they are few and far between and very ineptly placed. It is a little disturbing to find reproductions of salpingograms in the midst of a discussion on hernia.

These are relatively minor faults in a first-class production—whose field of usefulness should certainly extend far beyond the confines of the general practitioner and resident to whom it is dedicated.

Demonstrations of Physical Signs in Clinical Surgery. By HAMILTON BAILEY, F.R.C.S. (Eng.), F.A.C.S., F.I.C.S., F.R.S.E., Surgeon and Surgeon-in-Charge of the Genito-urinary Department, Royal Northern Hospital, London; etc. Eleventh edition. Issued in Four Parts. $5\frac{5}{8} \times 8\frac{3}{4}$ in. Part IV, Pp. 305–426 + iv, with 137 illustrations, a large number in colour. 1949. Bristol: John Wright & Sons Ltd. 8s. 6d. each part. Bound volume, 34s.

THE issue of Part IV completes the eleventh edition of this volume. As in the other parts, the illustrations are both profuse and clear and the text is used to supplement them. It is surprising to find no mention of the elevation of the testis in the scrotum which occurs in the condition

of torsion; it is a point of some importance in differential diagnosis. Nor can we find any allusion to disk lesions either in the differential diagnosis of sciatica or for the signs they produce.

The book can now be obtained in one bound volume of convenient size and moderate price. It is a tribute to both author and publisher and will remain one of the most useful adjuncts to the student and teacher of clinical surgery.

Atlas of Peripheral Nerve Injuries. By WILLIAM R. LYONS, Ph.D., Associate Professor of Anatomy, University of California Medical School, and BARNES WOODHALL, M.D., Professor of Neurosurgery, Duke Medical School. 10 × 12¾ in. Pp. 339, with 135 illustrations, 31 in colour. 1949. Philadelphia and London: W. B. Saunders Co. 80s.

THIS Atlas is concerned almost exclusively with the histological appearances of damaged nerves and is, without question, the best photomicrographic collection of its kind that has yet appeared. It reflects the great advances that have been made in staining methods and in photography. There are so many pictures that they indeed divert attention from the excellent text, which is alone sufficient to make the book worth while. Dr. Lyons is an anatomist, Dr. Woodhall a neurosurgeon with very considerable war-time experience of nerve injuries, and between them they succeed in showing the relevance of the morbid anatomical changes in damaged nerves to the problems presented by the surgery of their repair. The material has been drawn from many sources and has been pieced together in such a way as to present remarkably coherent accounts of what happens when a nerve is damaged. For example, one is shown the harmful changes in the peripheral stump that take place progressively with lengthening periods of denervation. The practical conclusion is that the sooner a divided nerve is repaired the better. But it is also demonstrated that the theoretical ideal, namely, primary suture, does not work out well in practice, for there may be interstitial damage of the nerve stumps, especially after war wounds, that cannot be detected soon after injury; it reveals itself later as more or less extensive intraneural scarring that often completely blocks the path of regenerating axons. The reader will find almost equally illuminating sections on injuries in continuity, traction lesions, and the fate of nerves that have been wrapped in various materials after suture.

Altogether this is a very good book, and a fitting tribute not only to the authors but to the many American surgeons who in the hurly-burly of war found time to learn something from the injuries they were called upon to treat. It is, perhaps, heavy going for the surgeon unfamiliar with the histology of the peripheral nervous system, but if he cares anything at all about the proper handling of nerve injuries he will at least make the attempt to read it.

Army Foot Survey. An Investigation of Foot Ailments in Canadian Soldiers. By Colonel R. I. HARRIS, M.C., R.C.A.M.C., and Major T. BEATH, R.C.A.M.C. 8½ × 11 in. Pp. 416, with 114 illustrations and many charts, etc. In typescript. 1949. Ottawa: National Research Council of Canada.

THE authors, in the Preface, state: "In spite of Napoleon's aphorism an Army marches, not upon its stomach, but upon its feet; even a mechanized army demands much of its soldiers. They must be able on occasion to march far and fast; to slog through mud and snow and to hold defensive positions unhampered by incapacitating foot problems. . . . There is need for information upon (1) the extent of foot disablement among the male population of enlistment age, (2) the various types of foot disabilities and their relative incidence, (3) the significance of various foot lesions in terms of function, (4) what foot problems may develop during military training, and (5) what can be done to minimize wastage from foot disabilities by preventing their occurrence or by salvage after they have developed."

This monumental report will surely be the 'classic' which will be accepted by all Army authorities in future as their guide and vade-mecum in assessing the foot disability and marching capacity both before and after training of their recruits.

The first volume is the text-book part and the second is a series of appendixes explaining the methods of approach and their interpretation.

The first volume, after explaining the Canadian Army organization in relation to foot problems and how these present themselves during the various stages of training, gives the plan of study and then the data collected, and this is followed by a detailed discussion of this data.

The appendixes give the minute detail of the data obtained and method of recording it and the conclusions drawn, e.g., the technique of obtaining standardized radiographs and the method of studying them.

The final appendixes deal with the miscellaneous observation and suggested revisions of physical standards, and, last but not least, the subject of 'Lasts' or suggestions for improving the footwear of the soldier in the light of the researches made and conclusions drawn from them. The honest endeavour to implement these conclusions is summed up in the words of one of the authors: "I wish to put down some suggestions for improving shoes and boots, I wish it to be understood that they are untried, but are suggestions I think should be tried.

"I am mixing in suggestions which I think are good with suggestions which I think are perhaps doubtfully good, but the latter should be considered in making new designs."

It is interesting to read a report such as this in an age of rationalization and mass production.

Medicine in all its branches, and particularly in surgery, must remain a craft, and each case must be considered on its own merits, and the treatment to afford relief must be a separate problem.

The report convinces one that there must be some standard method of investigation and recording so that the work can proceed at many centres at the same time, thus providing that an inclusive overall view can be taken when attempting to draw definite conclusions as to the magnitude of the problem.

The authors are to be congratulated on the clarity of their exposition and on the immense amount of work that must have been involved in carrying out such a research.

The book should be available in every orthopædic department and foot clinic throughout the Empire and Dominions. It might be easy for some orthopædic surgeons to quibble over some small details, but by and large they must admit, when they have perused it, that this report gives them a firm base from which to proceed for any future research they may wish to make on the disabilities of the foot.

BOOK NOTICES

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest to our readers.]

Lehrbuch der Massage und manuellen Gymnastik, unter besonderer Berücksichtigung der Sportmassage. By Professor W. THOMSEN, Frankfurt-a.-M. Second edition. 6¾ × 9½ in. Pp. 237 + xi, with 91 illustrations. 1949. Stuttgart: Georg Thieme. Bound, DM. 16.50.

Gemeinsame Erkrankungen aus der inneren Medizin und Chirurgie. By Dr. WALTHER KANERT, Herborn, and Dr. KURT AUGUST KOELSCH, Magdeburg. 6¾ × 9½ in. Pp. 500 + xx, with 49 illustrations. 1949. Stuttgart: Georg Thieme. Bound, DM. 35.

Darmbrand (Enteritis Necroticans). By K. HANSEN, E. JECKELN, J. JOCHIMS, A. LEZIUS, H. MEYER-BURGDORFF, and F. SCHÜTZ, Lübeck. 6¾ × 9½ in. Pp. 212 + iv, with 103 illustrations, some coloured. 1949. Stuttgart: Georg Thieme. Bound, DM. 25.

Untersuchungen über den lumbalen und cervikalen Wirbelbandscheibenvorfall. By Professor F. REISCHAUER, Essen. 6¾ × 9½ in. Pp. 88 + viii, with 25 illustrations. 1949. Stuttgart: Georg. Thieme. Paper, DM. 9.60.

Das Adenocarcinom des Collum uteri. Histologische, klinische und therapeutische Ergebnisse. By Doz. Dr. HANS LIMBURG and Dr. KLAUS THOMSEN. 6¾ × 9½ in. Pp. 87, with 39 illustrations. 1949. Stuttgart: Georg Thieme. Paper, DM. 6.80.

Medullary Nailing of Küntscher. By LORENZ BÖHLER, M.D., Director of the Hospital for Accidents in Vienna; Professor of Surgery at the University of Vienna. First English edition, revised by the author. Translated from the Eleventh German edition by HANS TRETTER, M.D., Trenton, N.J. 6⅞ × 10 in. Pp. 386 + xi, with 1261 illustrations. 1949. London: Baillière, Tindall & Cox. 38s. 6d.

Modern Practice in Anæsthesia, 1919. Edited by FRANKIS T. EVANS, M.B., B.S., F.F.A.R.C.S., D.A., Honorary Anæsthetist, St. Bartholomew's Hospital, London. 6⅝ × 9⅝ in. Pp. 606 + xx, with 227 illustrations. 1949. London: Butterworth & Co. (Publishers) Ltd. 50s.

Varicose Veins. By R. ROWDEN FOOTE, London. 6⅝ × 9⅝ in. Pp. 226 + xvi, with 181 illustrations and 3 coloured plates. 1949. London: Butterworth & Co. (Publishers) Ltd. 32s. 6d.

De Bouw en Ontwikkeling van het mediastinale Bindweefsel. By WILLIAM ABRAHAM BAX, Leiden. 6¼ × 9½ in. Pp. 208 + x, with 44 illustrations. 1949. Leiden: Eduard Ijdo N.V.

Varices del Miembro Inferior. By ALBERTO E. LAURENCE, Buenos Aires. 7 × 10⅝ in. Pp. 208, with 69 illustrations. 1949. Buenos Aires: El Ateneo. Price not stated.

The Portraiture of William Harvey. By GEOFFREY KEYNES, M.A., M.D. (Cantab.), F.R.C.S. (Eng.), Emeritus Surgeon, St. Bartholomew's Hospital. 7¼ × 9¾ in. Pp. 42 + viii, with 32 plates. 1949. London: The Royal College of Surgeons of England.

Hand und Arm. Ihre Erkrankungen und deren Behandlung. By Prof. Dr. med. GEORG HOHMANN, München. 6½ × 10 in. Pp. 272 + viii, with 199 illustrations. 1949. Munich: J. F. Bergmann. DM. 21.

Grundlagen der funktionellen urologischen Röntgendiagnostik. By Dr. med. habil. WALTER PFEIFER, Sandersbuck. 6½ × 9½ in. Pp. 88, with 48 illustrations. 1949. Stuttgart: Georg Thieme. Paper, DM. 9.60.

Operative Surgery. By FREDERICK C. HILL, B.A., M.S., M.D., Associate Professor of Surgery, The Creighton University School of Medicine, Omaha, Nebraska. With a Foreword by CHARLES W. MAYO. 6 × 9¼ in. Pp. 698 + xiv, with 255 illustrations. 1949. New York: Oxford University Press. 63s.

Experimental Surgery. By J. MARKOWITZ, M.B. (Tor.), Ph.D., M.S. (Minn.). Associate Professor of Physiology, University of Toronto. With a Foreword by C. F. SCHOTTLAUER. Second edition. 6 × 9 in. Pp. 546 + xiv, with 330 illustrations. 1949. London: Baillière, Tindall & Cox. 40s.

Das chronische cystische Hydrom der Dura in seinen Beziehungen zum sog. chronischen traumatischen subduralen Hämatom und zur Pachymeningitis hämorrhagia interna in Lichte der Relationspathologie. By Doz. Dr. med. habil. ALFRED BANNWARTH, München. 6½ × 9½ in. Pp. 57 + viii, with 19 illustrations. 1949. Stuttgart: Georg Thieme. Paper, DM. 9.60.

An Introduction to Clinical Surgery. By CHARLES F. M. SAINT, C.B.E., M.D., M.S., F.R.C.S. (Eng.), Hon. F.R.A.C.S., Hon. F.G.S.S., formerly Professor of Surgery, University of Cape Town. Second edition. 9¾ × 6 in. Pp. 384 + viii, with 373 illustrations. 1949. London: H. K. Lewis & Co. Ltd. 45s. net.

Cirugia de la Glandula Tiroides. Anatomia Quirurgica del Cuello. By DIEGO E. ZAVALETA. 10¼ × 7 in. Pp. 154 + xii, with 58 illustrations. 1949. Buenos Aires: Ediciones Impaglione. m.$n. 40.

Confrontations radio-anatomo-cliniques. Edited by M. CHIRAY, R. A. GUTMANN, and J. SÉNÈQUE. Fasc. III. 13 × 10 in. Pp. 80, with 150 illustrations. 1949. Paris: Masson et Cie. 1000 fr.

L'Asepsie. Etude documentaire et Bibliographie sur L'Asepsie chirurgicale. By MAURICE TERRILLON. 7½ × 5½ in. Pp. 356, illustrated. 1949. Paris: Masson et Cie. 650 fr.; De luxe edition 900 fr.

X-ray Diagnosis. For Clinical Students and Practitioners. By G. SIMON, M.D., B.Ch. (Cantab.), F.F.R., Assistant Radiologist at St. Bartholomew's Hospital and Assistant Director of the X-ray Department, The Brompton Hospital. 9½ × 7¼ in. Pp. 208 + x, with 180 illustrations. 1949. Cambridge: W. Heffer & Sons Ltd. 20s. net.

Cystoscopy and Urography. By J. B. MACALPINE, D.Sc., F.R.C.S., lately Honorary Surgeon and Surgeon-in-Charge of the Genito-urinary Department, Salford Royal Hospital; Consulting Urologist, Christie Hospital and Holt Radium Institute, Manchester; etc. Third edition. 9 × 6 in. Pp. 554 + 16, with 338 illustrations in the text and 15 coloured plates. 1949. Bristol: John Wright & Sons Ltd. 63s.

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